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RESULTS OF SURGICAL THERAPY FOR FOCAL EPILEPTIC SEIZURES*

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A. Introduction

From time to time a surgeon should scrutinize his own results and make that scrutiny available to others. The purpose of this study is to determine the results of cortical excision in the treatment of epileptics over a six-year period from 1945 through 1950 inclusive; to summarize briefly two earlier reports of previous surgical therapy (Penfield and Erickson, 1941; Penfield and Steelman, 1947), and so to provide adequate information upon which the success of the procedure in our hands can be judged. The follow-up analysis of this series was carried out by the junior author, as in the previous studies.

In these three successive series which extend over a period of 20 years there is, of course, a gradual evolution of method which has served to enlarge the field of operation. An attempt has been made, in this present paper, to analyse the reasons for the success or failure of operation, and to indicate those features which have prognostic value. The physical and, particularly, the mental effects of cortical removal will be referred to superficially here and given more exhaustive study in later publications.

The senior author turned his attention quite early to the problem of focal epilepsy by studying the cytology of the brain and the healing of brain scars. This was followed by a visit to Breslau, to the Clinic of Otfried Foerster who had, at that time, carried out radical excision of atrophic brain lesions in a small series of patients who suffered from focal epilepsy. The excised tissue was studied by all available methods (Foerster and Penfield, 1930) but the nature of the irritant which makes areas of gray matter become epileptogenic was not discovered. Unfortunately, the nature of this irritant is still the unsolved riddle, the secret of a sphinx that seems to smile at our everchanging therapy.

Much has happened in this field in 25 years (Penfield and Jasper, 1954). We have electroencephalography now and better anticonvulsive drugs. We have learned the localizing value of epileptic patterns and can detect the site of initial discharge in brain stem epilepsy as well as cortical seizures. We can recognize temporal lobe epilepsy and understand the mechanism of its production when birth compression is its cause.

And yet, since we are ignorant of the essential cause, therapy must continue to be judged empirically.

B. Rationale

Radical operation depends, for its rationale, upon the conception, elaborated repeatedly by Hughlings Jackson, that every epileptic seizure begins with ganglionic discharge in some area of gray matter. The aim of surgical excision is to remove the focal area in which epileptogenic discharges originate and thus put an end to the seizures.

This would be as simple as it is logical if the focal area were invariably discrete and if the surgeon could be sure to leave behind him normal cortex in a normal postoperative state. But completely discrete lesions are rare and the brain from which a part has been removed cannot be called normal, however great the effort to

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avoid scarring and to prevent alteration in the circulation of remaining gyri.

Sir Victor Horsley (1886) introduced the technique of subpial removal of cortical convolutions, and the technique is still in use. When convolutions are removed thus by suction, leaving a bed of white matter, and the circulation of the pial banks of the surrounding convolutions is spared successfully, there is little or no scarring. Electroencephalography at three weeks after operation may give little or no evidence of cortical abnormality. The difficulty, however, is to remove all of the epileptogenic cortex and that only.

An epileptogenic focus is not a point. It is a zone of cortex. This may be as small as a single shrivelled gyrus or it may consist in a group of somewhat atrophic gyri. When the obviously abnormal area has been removed there may remain hidden areas of abnormal cortex capable of carrying on the spontaneous discharge.

Habitual seizures rarely, if ever, begin at the time of brain injury. There is a ripening period of months or years that follows the initial insult. On the other hand, in many cases seizures never develop after brain injury.

Many patients have traumatic injuries of the brain without developing subsequent cerebral seizures. Many infants suffer acute anoxæmia of some area of cortex and consequently go through life with areas of focal cortical atrophy, large or small, without having to face the "curse" of epilepsy. On the other hand, many other such individuals do suffer from attacks because such abnormal areas of brain do become epileptogenic. Furthermore, a patient who is epileptic may have a large lesion of one hemisphere and yet seizures may seem to arise in only one area of injured cortex and not other areas of injured cortex.

C. Localization of the Epileptogenic Focus

The focus is that area in which the periodic outbreak of discharge seems to originate, the outbreak that gives rise to the initial phenomenon of the habitual attack. It is the area in which electrical potentials are highest between attacks but the extent of epileptogenic cortex may be considerably greater than these findings would suggest.

The position of the focus and of the abnormal epileptogenic cortex may be determined as follows:

1. Sometimes the nature of the initial phenomenon (sensory, motor or psychical) betrays the position of initial discharge. Thus the *ictal pattern* provides initial localizing guidance.

2. *Electroencephalography* may detect abnormal electrical potentials at the site of the pacemaker between attacks, and an enormous augmentation of such discharges at the time of attacks. When the recording electrode is placed directly upon such an area during operation, or when the scalp electrode is over a superficial cortical focus before operation, discharges of high potential and short duration ("spikes") are usually recorded. If the electrode is at a distance the disturbance may appear in the form of sharp waves or abnormal alterations in brain wave rhythms. At the onset of a seizure these interictal spike discharges become multiple.*

3. Electrical *stimulation* of the exposed brain may reproduce the initial phenomenon of the patient's habitual minor seizures and this may be taken as confirmatory evidence of the general position of the focus.

4. At the time of operation, *inspection* of the exposed brain often leads the surgeon to a presumptive identification of the epileptogenic focus. It can never be more than presumptive, for identification depends on the information just described. On the other hand, when the cortex is completely normal its removal gives little or no hope of a successful therapeutic result unless that removal serves to give access to initially hidden areas of objectively abnormal gray matter.

D. The Epileptogenic Lesion†

If the neurosurgeon finds that he is dealing with a "space-occupying" lesion, such as an encapsulated tumour, the focus is usually discovered in atrophic gyri nearby, gyri which have been narrowed by the progressive pressure of the neoplasm.

If a brain cyst is found, or an area of complete destruction such as that produced by intra-

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*In certain areas of the cortex these epileptogenic discharges are sometimes associated with a general suppression of electrographic activity. (See Penfield and Jasper, 1954.)

†The epileptogenic lesion may be defined as that area of gray matter in which spontaneous epileptogenic discharges arise. Tumours, cysts, vessel abnormalities, areas of destruction, adhesions—these may be associated findings, but the epileptogenic lesion is the gray matter that is still alive but has taken on this abnormal activity because of some pathological influence.

cerebral hæmorrhage or thrombosis, the focus may be found in the narrow gyri of tough consistency that border the cyst or the area of destruction. In such cases numerous gyri of this type may be discovered forming the boundary of the lesion, and we have learned by experience that such atrophic gyri should all be considered real or potential areas of origin and therefore should be removed when practicable.

If the abnormality of the cortex has been produced by birth compression the focus may be found in an area of small gyri (microgyria) or in shrivelled buried gyri.

Occasionally the surgeon may not encounter abnormal gray matter of the type that he considers could be epileptogenic until after he has made a partial removal guided by other evidence. Then he comes upon a zone of cortex that is sclerosed, yellowish, atrophic, tough. This may be on the undersurface of a lobe, as in incisural sclerosis of the undersurface of the temporal lobe. There may be atrophic gyri deep in the interhemispheral sagittal fissure.

The electrocorticograph is of increasing importance at the operating table before excision of convolutions and also as a check-up immediately afterward, and a help in deciding whether further removal is indicated and where. Spike discharges and sharp waves may appear on the border of an initially removed area where they were not observed before. This is not an evidence of traumatic effect but indicates that some area of epileptogenic cortex has been overlooked.

The "diviner's rod" is said to be used to find water. The electrograph detects epileptogenic abnormality, whether on the surface or hidden, and the surgeon must find it with a minimum of damage. Removal of normal convolutions will not bring therapeutic success, and therefore electrographic evidence is not sufficient to justify excision of normal brain unless it leads to exposure of hidden abnormality.

After years of experience with the gross appearance and the microscopic structure of the gray matter of epileptogenic areas, it is possible to point out certain characteristic features.

In the total series of patients, 78 were found to have clearly atrophic lesions of the cerebral cortex. Ten more had cerebral cysts with atrophy of surrounding gyri. There were 45 examples of meningocerebral cicatrix. Such a scar results from cerebral laceration or local abscess formation and drainage which produces a central core of connective tissue, densely adherent to the dura. This central mixed scar is surrounded by atrophy and gliosis of cerebral tissue.

In 25 cases there were cortical areas of microgyri, such as are produced by birth compression, obvious to inspection on the surface.* In the ten examples of cerebral cyst mentioned above, there were microgyri around the margins of each cyst.

Incisural sclerosis of the hippocampal and uncal area is the most frequently encountered pathological entity. The manner of production of this lesion by herniation of temporal lobe through the incisura of the tentorium with arterial compression is described below under the heading of Birth Lesions.

In the convolutions which form the epileptogenic focus a varying degree of atrophy is often evident on inspection. The consistency is increased so that the tissue enters the aspirator (sucker) used for cortical removal less readily. Sometimes it is so tough and rubbery that it must be removed by sharp dissection.

In this series of cortical excisions the operator was prepared to say that there was gross abnormality in the tissue removed in all but eight cases. Presumably the removal in those eight cases was made entirely on the evidence provided by electrocorticography and electrical stimulation. It may well be significant, in passing, that only two of the eight patients were eventually placed in the success groups of the follow-up analysis.

Histologically, the toughness of epileptogenic cortex seems to be due to the change of protoplasmic astrocytes into fibrous astrocytes in the gray matter and the over-all increase in these cells. Careful microscopical study also often reveals the presence of minute foci of cortical destruction, either acute or long standing, as though at scattered points the circulation had become progressively inadequate even many years after the initial injury to the brain.

In general, it might be concluded that one feature is common to all of these cortical epileptogenic lesions, i.e., a slight persistent inadequacy of circulation. This could cause the patches of acute swelling of oligodendroglia which are found, the fibrous gliosis of astro-

*When such gyri were encountered deep to the surface, it was often difficult to see the size of the gyri although the toughness of the tissue removed was obvious. In such a position the operator did not call them microgyri in his operative note.

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cytes, the occasional dropping out of ganglion cells, and the minute islands of phagocytosis. These common features are found in convolutions compressed by an expanding lesion, but they are also found in areas which had been partly destroyed by acute ischæmia years before the onset of the seizures.

It remains to be shown by further histological study, or by other methods of analysis, wherein the non-epileptogenic atrophic gyrus differs from the epileptogenic atrophic gyrus. It remains to be determined whether this hypothetical inadequacy of circulation is really present between attacks and whether it may thus be related to the cause of continuing interictal discharge, or whether it is a purely passive expression of the excessive increase in the metabolic demands made upon the tissue during a seizure.

It remains to be shown, also, just what the nature of the local biochemical peculiarity may be in the epileptogenic cortical focus. The work on this subject which has been carried out in the Neurochemistry Laboratory of our associate, Dr. K. A. C. Elliott, has been summarized by him in the following personal communication:

> Biochemical studies have shown an anomaly in the metabolism of acetylcholine in human focal epileptogenic tissue (Tower and Elliott, 1952, also 1953). Normal brain tissue, when incubated *in vitro* under appropriate conditions, produces free acetylcholine and also increases its content of the bound form of acetylcholine. Epileptogenic tissue fails to produce this bound acetylcholine. The same failure is found in cortex from animals suffering repeated seizures following the administration of methionine sulfoximine. This anomaly may be the basis for the tendency of the tissue to initiate seizure activity *in vivo*. Epileptogenic tissue *in vivo* may tend to produce free, physiologically active acetylcholine at times when normal brain would store the acetylcholine in the inactive bound form.
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> The nature of the anomaly is not yet clear from the biochemical standpoint. The same defect appears in normal tissue *in vitro* under conditions of partial anoxia. The production of bound acetylcholine can be restored to normal by the presence *in vitro* of glutamine or asparagine. The production of bound acetylcholine is found to be greatly increased in tissue from animals treated with certain narcotics and anticonvulsants. Evidently the changes in production of bound acetylcholine depend in a complex manner on the condition of metabolic systems. Earlier work (Elliott and Penfield, 1948) had shown that there is no simple relation between respiratory or glycolytic capacity and epileptogenic activity.

E. THE NEUROSURGEON'S TASK

This field is a difficult one. There are as yet few rules of routine procedure. Success must depend upon balanced clinical judgment.

The neurosurgeon who would enter the field must learn to identify the abnormal cortex in which epileptic discharges are apt to originate by means of clinical, physiological, and electrical analysis. He must carry out his excisions with a minimum of disturbance of the circulation of the remaining gyri. He must balance the chance of freeing his patient from seizures against the risks and functional losses that may be associated with ablation.

Thus it is evident that conclusion as to location of an epileptogenic focus must depend upon all of the available information: (1) seizure pattern; (2) electroencephalography before and electrocorticography during operation; (3) electrical stimulation of cortex under local anæsthesia; (4) the appearance and consistency of the cortex.

There are other important aids—the help of a neurologist who understands all forms of modern medical treatment and also repeated consultation with a wise electroencephalographer who understands the total problem. When operation is being considered it is almost always possible to identify, by means of the E.E.G., those cases in which the originating focus of epileptic discharge is subcortical. Thus, with rare exceptions, it is now possible to avoid negative exploratory craniotomies.

F. MATERIAL

During a six-year period (1945-1950 inclusive) 234 patients were treated by craniotomy for the relief of epileptic seizures. This series will be compared in the final discussion with the 76 similar cases analyzed for the preceding six years (1939-1944 inclusive). None of the patients included had an intracranial tumour. During the same period approximately 80 additional patients who actually had such tumours came to operation with the primary complaint of seizures rather than pressure. They were operated upon also—but they will not be considered in this study.*

All of these patients reported here were under the care of one of the authors (W.P.) and were operated upon by him or by one of his neurosurgical assistants.†

*Still other patients whose seizures were obviously secondary to intracranial tumour were admitted to the Montreal Neurological Institute during this period but will not be included in this analysis. Most of them were obviously suffering from increase of intracranial pressure and many of them were operated upon by our associates, Dr. William Cone and Dr. Arthur Elvidge.

†Drs. Theodore Rasmussen, Keasley Welch, Frank O'Brien, John Hanbery and Maitland Baldwin contributed each in turn to the study and the care of these patients and we owe them an obvious debt of gratitude.

1. *Follow-up Method*

Our follow-up method was as follows: An attempt was made to discover the fate of every patient operated upon in the six-year period 1945-1950 inclusive. The follow-up was continued at least to the end of 1951, so that there is a minimum follow-up of one year from the date of operation and a maximum of seven years. In most instances, the files consisting of inpatient and outpatient records and correspondence did not contain all the information required, and such patients received a questionnaire form to be completed and returned.

Approximately 190 patients filled out the form and returned it. This questionnaire was made as simple as possible with the intention that a letter would be sent later asking for answers to specific questions if further information should be required. The patient's relatives were encouraged by the questionnaire to write a letter to add their opinions to that of the patient, and in about half of the cases they did so. In some instances it was necessary to write a supplementary letter to the patient's own doctor, for elaboration of ambiguous replies. A relatively small number of patients lived in Montreal or were able to return for examination during 1952. Fuller details are thus available for these cases.

In spite of the inevitable difficulty of securing exact information by correspondence, the summaries in the following pages seem to provide material adequate for assessment of the value of cortical excision as used for the treatment of focal epileptic seizures during this six-year period.

2. *Sex Incidence*

Table I shows the sex distribution in the group of 234 patients with epilepsy due to non-neoplastic lesions of the brain. It shows, also, the relationship of this distribution to probable cause of seizures.

TABLE I.

SEX INCIDENCE AND RELATIONSHIP TO CAUSE

Etiology	*Male*	*Female*
Birth lesions	57	27
Post-traumatic	61	19
Miscellaneous	35	35
Total cases (234)	153 — 65%	81 — 35%

Males outnumber females as a whole, 65% to 35%. In the post-traumatic epilepsy group they outnumber the females 3 to 1. This might be expected, as the incidence of head injury is generally greater in males, and especially so in our series which includes servicemen who have had war wounds of the head. However, it was unexpected that males should have exceeded females 2 to 1 when the cause was birth injury or birth anoxia. Holt (1940) gives the average circumference of the head of the newborn infant as 13.9 inches for the male against 13.6 inches for the female, a rather small difference in size to account for the difference in incidence of brain injury.

The cause in each case was frequently a matter of difficult decision. But, after operation and microscopical study of excised tissue, a presumptive conclusion has been made that must be correct in the great majority of instances. This will be discussed further.

3. *Family History*

In all but 10 of the 234 cases in this series our hospital records gave specific information regarding the occurrence of seizures in other members of the family. Eleven near relatives of 10 patients have been, or are, afflicted with epilepsy. Thus, 4.4% of the patients have a family history of some convulsive disorder. More distant relatives, of an additional seven patients, had had one or more convulsions. As the average number of near relatives, calculated from the Canadian Census figures, is about five for each patient, the incidence of epilepsy in the total number of near relatives is thus estimated to be below 1%.

This approximates the incidence of 0.9% for all forms of epilepsy among Americans drafted for service in the U.S. Army during the Second World War. Alström (1950), referring to epilepsy of known etiology only, in Sweden, gives the incidence of epileptic relatives as 1.1%.

Therefore, it seems fair to conclude that hereditary tendency may be ruled out as a factor in the development of focal cortical seizures among the patients in this series.

A misleading example of apparent hereditary tendency might be cited here.

Two sisters (B.S. and M.N.) suffered from seizures due, in each case, to a temporal lobe focus. Each was operated upon and, in each case, sclerosis of the mesial zone of the temporal lobe was found and the area removed. The sclerosis was clearly due to temporal herniation through the incisura of the tentorium at the time of birth. The significant factor in the production of "incisural sclerosis" was the pelvis of the mother through which each child came into the world, rather than a common inheritance of a tendency to epilepsy.

There was a history of mental or personality disorder among relatives of 10 patients. It is interesting that seven of these were in families in which epilepsy was also reported among the relatives.

G. ETIOLOGY

It was stated above that there are common features which may be recognized by gross and microscopical examination in the excised epileptogenic tissue. But it is obvious that these

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common pathological changes are produced in a great variety of ways by many pathological agents. These agents are summarized in Table II.

TABLE II.

CAUSE OF FOCAL CORTICAL SEIZURES

	Known	*Probable*	*Total*
1. Birth lesions	67	17	84 — 36%
2. Postnatal traumatic lesions.	70	10	80 — 34%
3. Local suppuration	13	0	13 — 5%
4. Cortical thrombophlebitis	6	4	10 — 4%
5. Angiomas	7	0	7 — 3%
6. Hæmangioma calcificans	4	0	4 — 2%
7. Miscellaneous			18 — 8%
8. Lesions of uncertain etiology			18 — 8%
		Total cases	234 — 100%

This table shows the nature of the cerebral lesion or the mechanism of its production, so far as it could be ascertained from the history of the patient's illness, the findings of special examinations, and the gross features of the lesion discovered at operation or its later investigation by histological methods.

The table illustrates the fact that about one-third of all cases fall into a group in which birth injury or anoxæmia was assigned as the cause. In a second group of about one-third of the cases the cause was postnatal trauma. These and the smaller groups are considered below separately for purposes of analysis and comparison. The cases in which there was good presumptive but not certain evidence as to cause are placed under the heading "Probable" in Table II.

1. *Birth Lesions*

An abnormality that was produced in the brain during birth we have called a birth lesion. There may have been actual trauma by instruments, or unequal cerebral anoxia, intracranial hæmorrhage or herniation of temporal cortex through the tentorial opening. One of these, or a combination of them, may be the actual cause of the abnormality, but it seems likely that cortical ischæmia or anoxia most often plays the important role.

It is, to some extent, the area of brain injured at birth which determines whether or not the child's history will give evidence of it. A lesion of the central region produces a defect in the use of arm and leg on one side that parents usually detect within the first six months. Failure of the infant to suck well during the first few weeks probably points to diffuse injury or perhaps compression of the brain stem.

But a lesion of the temporal lobe may be produced by head compression during birth without resulting in any defect in the child's behaviour which even the most expert pædiatrician can detect.

Earle, Baldwin and Penfield (1953) made a pathological study of 157 consecutive cases in which seizures originated in the temporal lobe. Many of these cases are included in this follow-up study. In 100 of these cases (63%) it was concluded that anoxæmia or compression at the time of birth had been the actual cause of lesion and seizures. Smallness of the middle cranial chamber, as shown by x-ray, is sometimes found and may be taken as strong confirmatory evidence that the lesion dates from birth or early infancy.

The pathological lesion thus produced consisted in sclerosis of the infero-mesial zone of the temporal lobe. This they showed could be produced by herniation of temporal convolutions through the incisura of the tentorium into the posterior fossa, thus probably producing temporary compression of the arteries which cross the free edge of the tentorium to supply the uncus, hippocampal gyrus and adjacent cortex.

This pathological change they designated as *incisural sclerosis*. At the time of operation this lesion can be recognized easily by the colour and consistency of the tissue (Penfield and Baldwin, 1952). Consequently, when it is encountered and when no other obvious cause is discovered, and when the seizures are arising in the area, the conclusion may be drawn that birth compression is the real cause, even though no abnormality was reported by obstetrician or parents at the time of birth.

Febrile convulsions of infancy and childhood: The fact that the first convulsion may have occurred at the time of a febrile illness early in life does not necessarily throw any light on the nature of the cerebral lesion. We believe that the fever in such cases is most often a precipitating factor, rather than evidence of encephalitis. Thus, the child with an unsuspected atrophic lesion of the cortex may have his first attack due to the precipitating influence of a febrile illness, or even a mild head injury. The objective findings at operation bear out this

belief.* In Table II there are 84 examples of birth lesion, and in 28 of these cases there was history of an early convulsion during a febrile illness early in life.

TABLE III.

TIME OF ONSET OF RECURRING SEIZURES DUE TO A BIRTH LESION — 67 CASES

Age at onset	*Lesion in temporal lobe*	*Lesion elsewhere*
0 to 5 years	1 — 4%	20 — 46%
5 to 10 years	8 — 34%	9 — 21%
10 to 20 years	10 — 42%	13 — 31%
20 to 55 years	5 — 20%	1 — 2%
Totals.....	24	43

Onset.—Table III shows the age of onset of the habitual seizures following a cerebral birth lesion. Only the 67 cases in which the cause was certain, rather than presumptive, are chosen for this analysis. It is seen that temporal lobe seizures commence most often between the ages of 5 and 20 years, 78% in all. On the other hand, those affecting other parts of the brain become manifest in the first five years of life in almost half the patients, 48%. Another 40% appear between ages 5 and 20. The mean age of onset of convulsions is 13 years in the temporal lobe cases and six years in the others.

It has been pointed out above that the cause of the temporal lobe damage in birth injuries is evidently temporary vascular occlusion and ischæmia of infero-mesial temporal cortex. On the other hand, the lesions elsewhere are usually produced by direct trauma or hæmorrhage. Superficially placed temporal lobe foci may have originated from direct cerebral trauma, but these lesions are rare when compared with incisural sclerosis of the temporal lobe.

First-born.—The patient's position in the family has been noted in those cases where a birth injury is suspected as the cause of later convulsions. In the 53 case records where this evidence is available, it is found that there are 33 first children, 9 second, 6 third, 2 fourth, and 3 fifth or later. The Canadian Census figures for 1948 give the following proportions in the total population: 31% for first-born, 25% for second, 15% for third, 9% for fourth, and 18% for the remainder. If one can assume that the deaths in each group are in proportion to the total numbers in each, then first-born children are more commonly met with in the patients with focal epilepsy due to birth injury in our series than in the general population.*

2. Traumatic Lesions (Postnatal)

In many instances, it was obvious that a local lesion of the brain had been produced at the time of trauma. This was especially so in the cases of penetrating injury and depressed fracture of the skull. On the other hand, when the patient had sustained a closed head injury without perforation of the dura by depressed bone fragments, the evidence was often not so clear. But the presence of a hemiparesis or sensory loss, or of an aphasia dating from the time of the injury, may be taken as an indication of a localized lesion of the brain. Unconsciousness, following trauma, indicates only the degree of severity of the trauma.

In this series we have not accepted trauma as the cause unless there was a localized epileptogenic lesion that seemed to be beneath the point of impact, or in a few cases in a position favoured as a site of contrecoup injury, such as the tips of the temporal lobes or tip and undersurface of the frontal lobes. In the case of direct or of contrecoup injury, it is expected that adhesions between dura and brain will be present if that trauma is to be accepted as cause.

In one case, for example, the epileptogenic focus proved to be an area of atrophic cortex with fine adhesions between it and the overlying dura. It lay directly beneath a scar in the scalp that marked the point of impact of a blow. The patient had no recollection of the event! Nevertheless, this was taken as adequate evidence of the traumatic nature of the original lesion.

In another case of so-called closed injury of the skull, a basal fracture in the middle fossa was found and there was a tear in the dura at the site of fracture. This had produced erosion of the bone at the site of the dural defect and there were dense, but localized, adhesions between dura and brain in the neighbourhood. The patient's epileptogenic lesion was on the

*It is of course also true that in groups 3 and 4 (Table II, cortical thrombophlebitis and localized suppuration) there were initial seizures at the time the lesion was acquired in the majority of cases. After such infections there followed a lapse of time (the ripening period) before the eventual onset of habitual cerebral seizures.

*No allowance has been made for the changing birth rates, and the population figures apply to all individuals. Furthermore, the case histories are more likely to contain reference to single children, i.e., first-born, than to the position of other patients in a fraternity. Both of these factors would tend to make the incidence in the patients under discussion appear proportionately larger. No correction is made for American and foreign origins.

undersurface of the temporal lobe, in the region of this abnormality.

Onset of post-traumatic seizures.—Table IV shows the interval between the postnatal trauma and the onset of habitual seizures, indicating a mean interval of about a year after penetrating head injury, and of slightly less than a year after head injury.*

TABLE IV.

TIME OF ONSET OF RECURRING SEIZURES DUE TO POSTNATAL TRAUMA

Interval after injury	*Dura penetrated*		*Dura not penetrated*	
Less than 1 year	21	50%	25	58%
1 to 5 years	11	26%	6	14%
5 to 10 years	8	19%	7	16%
Over 10 years	2	5%	2	5%
Interval uncertain	0		3	7%
	42		43	

The table is made up with regard to the time of onset of habitual recurring seizures. Five of these patients also had one or more seizures immediately following injury. In three of them the habitual attacks began at four, five and six months after the injury. In the case of one, the onset was delayed for one year, and in the remaining case for five years. Three other patients had seizures during the acute phase of injury, which could also be said to be the time of onset of their habitual seizures. That is to say, no attack-free interval could be recognized after the injury.

3. *Localized Suppuration*

Table II shows that there were 13 patients who suffered from some form of intracranial infection before the onset of their seizures, and in whom it appeared that the infection had a causal relationship to the attacks.

Seven of these patients had had a brain abscess previously treated by drainage. Four had a primary focus in the ear, and in one, although the intracranial abscess followed a tonsillectomy, an unrecognized focus of infection may have existed in the middle ear as well. One patient had an osteomyelitis of the frontal bone following scarlet fever, with later intracranial extension of the infection. The other patient, with a parieto-temporal abscess, had no detectable primary source of infection.

Onset.—The interval between the acute stages of the infection and the onset of seizures was 2 years in three cases, 3 years in two, and 5 and 10 years respectively in the other two. One additional patient had had a subdural abscess secondary to mastoiditis and developed habitual seizures 13 years later.

The five remaining patients had infected penetrating wounds of the brain and are considered with the traumatic cases with regard to the onset of their seizures in relation to the causal agent.

4. *Cortical Thrombophlebitis*

Ten patients were believed to have suffered a cortical venous thrombosis in infancy which had led to changes in the cortex, later to produce an epileptogenic focus. In most patients with such a history, as mentioned above, it appears that the febrile illness is only a precipitating factor when the brain has already been disturbed by a birth lesion, a very early trauma, or a vascular abnormality.

Before accepting cortical venous thrombosis as the probable cause of seizures, we have demanded evidence of brain damage at the time of the pyrexial illness. This evidence is usually hemiparesis, sensory loss, visual field defect, or aphasia which persisted for some days or even weeks, thus differing from the usual post-ictal neurological defect which ordinarily clears up in a shorter period of time. The distinction, however, must often remain in doubt.

5. *Angiomas*

Table II shows that there were seven angiomas in the series. These included three capillary hæmangiomas, one racemose arteriovenous communication, one cortical arteriovenous malformation and one capillary hæmangioma, previously excised in another clinic without cessation of seizures. In one of these cases calcification was present. In all of these cases the vascular abnormality was excised, together with the surrounding convolutions which formed the epileptogenic abnormality. In the seventh case a large racemose angioma of the right central region was investigated at operation but not excised for fear of producing a severe hemiplegia.

*Patients in this latter group are more likely to have been placed there if the interval between the trauma and the onset of seizures was short, and some truly post-traumatic epileptics may have been classed in the uncertain group because of the doubt as to causal relationship of trauma because of a long interval between trauma and seizures.

There were four more cases of a different type, described in the next section.

6. *Hæmangioma Calcificans*

There were four of these epileptogenic lesions. They constitute an interesting pathological group previously described by Penfield and Ward (1949). Degeneration is marked and vascularity is much reduced. In all of the four cases of this present series, the lesions were in the temporal lobe of one side or the other. This was true also of the previously reported cases.

7. *Miscellaneous*

Brief reference may be made to the 18 lesions included under this heading:

Postnatal ischæmia.—Two cases. In one case there had been arrest of respiration during general anæsthesia and in the other there had been a "narrow escape" from drowning.

Lead encephalopathy.—One. Ingestion of lead-containing paint was followed by coma of two days' duration with generalized convulsions. Habitual seizures began a few months later.

Cerebral malaria.—One. A little girl, aged 2, had malaria of the cerebral type. At 6 years habitual seizures appeared. At operation there was abnormality within the right Sylvian fissure suggesting an earlier inflammatory process, particularly over the island of Reil.

Tuberous sclerosis.—Two. In one case the condition was recognized only on postoperative microscopical study. Attacks were not arrested by excision.

In the other case, the nature of the pathological condition was clearly diagnosed before operation, but as the attacks were invariably of the same pattern and the E.E.G. focus was well localized, it was finally decided to yield to the parents' insistence and excise the epileptogenic area. It consisted of tough, rubbery gyri. Abnormality was obviously present elsewhere, but the result of operation is nevertheless excellent for the time being. The parents are delighted. During the three years since operation, attacks have stopped and the boy has improved greatly in intelligence and behaviour. He has improved in school work, and at home. He has learned his "catechism", a thing he had repeatedly failed to accomplish before operation! Since he took phenobarbital before and after operation, this is a good example of the effect of excision of "nociferous cortex" on the patient's intelligence and behaviour (Penfield, 1952).

Uræmia and acute nephritis.—One. At the age of 10 years, this condition, associated with hypertension, was thought to have produced the epileptogenic lesion of the brain. Focal motor attacks had been described at the time of his acute illness and recurred as habitual attacks nine years later.

Encephalitis.—Four. The diagnosis of encephalitis (probably due to virus infection) is always difficult to make with certainty. But examination of the case histories has led us to this conclusion in four cases. There was pleocytosis in the cerebrospinal fluid of two of the patients at the time of their initial illness, according to records from other hospitals. No bacteria were found, and the description of the illness was compatible with the diagnosis of encephalitis. The third patient, who was unconscious for a prolonged period during measles, was thought to have had a morbilliform encephalitis.

The fourth patient had had convulsions during a febrile illness associated with slight meningismus, but without any recorded changes in the cerebrospinal fluid. She was comatose for 12 days and developed a left hemiplegia at that time. We found evidence of moderate bilateral enlargement of the lateral ventricles in the pneumoencephalogram and, on operative exposure, extensive atrophy of the cortex was encountered.

Hæmorrhagic disease of the newborn.—One. During the first few days of life, this patient was noted to have petechial spots on her skin and mucous membranes. She also had melæna. On the third day, evidence of a cerebral lesion developed suddenly and physicians considered that the body had suffered a cerebral hæmorrhage. Years later, when craniotomy was carried out for relief of seizures, she was found to have a localized area of brain destruction, now filled in by an outpouching of the lateral ventricle. Hæmorrhagic diathesis of the newborn was taken as the cause of intracerebral hæmorrhage.

Brain tumour suspect.—Three. Included in the series are three patients who were suspected of having an intracranial tumour, but pathological examination of the biopsy specimens did not confirm this impression. These patients are still alive and show no evidence of spread of tumour inside the skull. For this reason they are retained in the non-neoplastic group but none had a cortical excision at the time of operation.

Meningitis.—Two. One patient, who had had a meningitis at the age of 7 years, developed seizures five years later. At craniotomy, 21 years after the onset of his seizures, the main abnormality found was thickening of the arachnoid over the exposed cortex. The second was a girl, whose attacks began six years after an illness diagnosed as meningitis, and who was found at craniotomy to have widespread but fine subdural adhesions. In addition, she had calcified granulomatous masses in both frontal lobes, one of which was removed at operation, the other only visualized by radiography. She was known to have had pulmonary tuberculosis in early life, and the granuloma was assumed to be tuberculous.

Arteriosclerosis.—One. Only one patient was over 55 years of age at the time of onset of his seizures. The exposed cortex in this case showed diffuse atrophic changes with slight yellowing of the fluid in the subarachnoid spaces. Our tentative conclusion was that the cortical abnormality responsible for his seizures was caused by sclerotic disease of the cerebral vessels. He had generalized arteriosclerosis without hypertension.

8. *Lesions of Uncertain Etiology*

In the remaining 18 patients, either the initial lesion is unknown, or two or more etiological factors could equally well be incriminated, so that the diagnosis is classed as uncertain.

9. *Etiological Significance of Age of Onset*

The time of onset* of the recurring seizures, in those cases in which there was clearly a lesion dating from birth, is collected in Table III. It is seen that the onset is considerably later in the temporal group. This may be due to the fact that direct trauma or laceration is apt to occur in other parts of the brain while incisural sclerosis is an ischæmic lesion. But the latency is, on the average, greater during infancy and childhood than later in life, regardless of location.

*In this section, time of onset of seizures is taken to be that time when the habitual attacks seem to have begun. A convulsion associated with a febrile illness of childhood or seizures occurring at the time of brain abscess or brain trauma are not considered as the signal of onset of recurring seizures.

This conclusion is borne out when Table IV is compared with Table III. In adult life half of the post-traumatic epileptics experience the onset of recurring attacks in less than one year. In contrast to this, half of the temporal cases due to an accident of birth begin around the age of puberty. Among adults there was little difference in the time required for "ripening" of the epileptogenic process when the penetrating injuries were compared with the non-penetrating (Table IV).

When the series of patients is re-examined from the point of view of age of onset of habitual seizures rather than the time after brain insult, Table V is produced. This shows that, of the 73

TABLE V.

AGE OF ONSET

ONSET AT AGE	0-2 yrs.	2-10	10-20	20-35	35-55
No. of cases	31	66	73	50	13
Due to birth lesion	58%	46%	34%	12%	31%
To postnatal trauma	13%	26%	37%	60%	54%
To miscellaneous	29%	28%	29%	28%	16%

patients whose seizures began between ages 10 and 20, for example, the cause was about equally distributed among birth, postnatal trauma and other causes.

TABLE VI.

CRANIOTOMIES FOR FOCAL EPILEPSY

Cases during 6-year period 1945-1950		234
Cortical excision, patients	219	
Negative explorations, patients	15	
Number of operations		268
Deaths		4
Case mortality		1.7%
Operation mortality		1.5%
Adequate follow-up		217

H. RESULTS OF OPERATION

Table VI enumerates the patients, the operations and the follow-up records to be included in the analysis of therapeutic results.

Among the 234 patients operated upon during the period 1945 to 1950 inclusive, 203 had one operation only in this time for the purpose of relieving epileptic seizures, 29 patients had two operations, one had three and one had four. Thus a total of 268 craniotomies were carried out with the intention of excising an epileptogenic focus. This does not include any operations for postoperative hæmorrhage, infection or repair of cranial defects. One operation performed in January 1951 is included, as this was the second on a patient during one admission, the first having been performed in December 1950.

Four of the patients had had a previous operation for epilepsy in the Montreal Neurological Institute and returned here during the period under study for a further procedure. One of these patients died postoperatively and the other three benefited only slightly by the second operation. All are included in the analysis.

Each of six patients had had one operation to relieve his epileptic seizures at another hospital before being seen at this Institute. Seizures had recurred, and the patients were seeking advice again (forever hopeful!). Only one of these was successfully relieved of the attacks, three were improved and the other two were unchanged. These also are included in the analysis.

Eleven patients who were operated upon for the first time between 1945 and 1950 have since returned, after 1950, for a second operation. The follow-up results in regard to these patients are accepted, as they were assessed before and not after the second operation.

Multiple operations during the six-year period under study, irrespective of the time interval between the craniotomies, have been analyzed as two-stage procedures and are thus only represented once for each patient.

There were four deaths in hospital after operation, so that the case mortality was 1.7% and the operative mortality 1.5%.

Table VI shows 219 patients who had excision of cerebral cortex that was considered to be epileptogenic and 15 craniotomies without excision. In the first group, 203 patients could be followed up satisfactorily and in the second, 14. In 11 cases there was inadequate follow-up, some have not been heard from since discharge from hospital, and the remainder were known to have had no seizures after operation during the short time that information was available, but since they could not be reached at the time of this final analysis they are not included here. Two patients died after discharge, one in a few weeks, the other in four months.

Assessment of results is possible, therefore, in 217 cases, 203 in which craniotomy was carried out and 14 in which exploration was negative.

TABLE VII.

POSTOPERATIVE SEIZURE RESULTS

Group				
4.	Perfect result	57	(28%)	Success — 45%
3.	Nearly perfect result	34	(17%)	
2.	Satisfactory result	40	(20%)	Good — 20%
1.	Slight improvement	37	(18%)	Failure — 35%
0.	No improvement	35	(17%)	
	Total cases followed up	203		

1. *Cortical Excisions*

The results of these excisions are outlined in Table VII. It should be borne in mind that these patients had all received what was considered adequate trial of the best medical therapy without reasonable success. The estimates of results, made after complete study by one of us (K.P.), are expressed in a form comparable to that used in the earlier case follow-up studies from this clinic.

For the purposes of this study, we have defined a seizure as an episode with one or more of the following features: ictal movement, lapse of consciousness or lapse of memory. Minor ictal sensations alone are not counted and neither are

alterations in consciousness or memory so brief as to pass unobserved by persons about the patient. These exclusions, which apply to a very few patients, seem reasonable since the surgical objective is to reduce the patients' social and economic handicap. The exact incidence of such subjective episodes, not included as seizures, will be described later.

Each of the five follow-up groups shown in Table VII may be defined as follows.

Group 4: No seizures since discharge from hospital.

Group 3: Not more than three seizures since discharge from hospital. Patients followed up for less than three years after operation must have no more than one attack a year if they are to be included in this group. Many of the patients in this group had one or two attacks shortly after discharge and subsequently none for the three, four, or five years up to the time of analysis. In such cases the result is gratifying and the patient probably considers himself cured, but we have not scored his result as perfect.

Group 2: A patient who is having six (or less) major seizures a year or six (or less) attacks of automatism, or less than 12 minor motor seizures a year, is classified in this group, provided this constitutes a significant improvement over the preoperative level of frequency.

Group 1: Seizures definitely less frequent or less severe than before operation.

Group 0: Little or no improvement in frequency or severity of seizures; in some cases worse than before operation.

In the discussion of results, group 4 will be called perfect, group 3 almost perfect and the two together will be rated as successful results. In group 2 the results may be considered satisfactory, but for groups 1 and 0 the operation will be considered to have failed from the point of view of control of seizures.

Medication.—It has been our invariable practice to forbid the use of all anticonvulsive drugs following operation, except phenobarbital. This drug is continued at 2 to 3 grains a day for adults. The tendency to postoperative attacks seems to decrease with the passage of time, and the patients in groups 3 and 4 usually stop all medication within a year or two.

Patients in lower success groups may find that the effectiveness of dilantin has been greatly increased by operation. They may consider this a most welcome change. Nevertheless any patient who feels that he must take dilantin after operation is recorded as a surgical failure.

In general, no patient has been accepted for operation in the first place until he has had extended preoperative trial of medication including adequate doses of phenobarbital and dilantin.

In conclusion, therefore, there are 57 patients who have had no attacks and 34 who had not over three attacks before apparent cessation of seizures.* If groups 4 and 3 are combined, operative therapy may be called a "success" in 45% of the total. In addition to this, in 20% of the cases there was what may be called a satisfactory result.

2. *Negative Explorations*

In 15 cases craniotomy was carried out but no cortical excision was deemed advisable. Nothing was done in addition to exploration except perhaps separation of adhesions or subtemporal decompression.

The records of these patients may serve as a control. None had a successful result. Of the 14 who could be followed up, none was in group 4 or 3, two were in group 2, two in group 1 and 10 in group 0.

The length of follow-up in this negative series was 6 years in one, 5 years in four, 4 years in three, 3 years in one, 2 years in four and 1 year in one.

In the series of cases previously published by Penfield and Erickson (1941) one patient was placed in group 4 as the result of negative exploration.† A second patient was placed in group 3, seven patients were put in group 2 and 26 in group 0.

In the series published by Penfield and Steelman (1947), no case of negative exploration was placed in group 4 or 3, but five were placed in group 2, two in group 1 and nine in group 0.

Thus it is seen that as the result of 65 negative explorations in the three series (470 patients in all), only two patients could be placed in the success groups (4 and 3). The cure of these two patients in a consecutive series of 65 negative craniotomies can hardly be considered as more than a chance variation.

3. *Partial Temporal Lobectomy*

It may be of interest to separate from the total 234 patients the 68 who were suffering from

*There were four patients who, because of excellent performance in subsequent years, were finally raised to group 3 from group 2. They did have more than three early seizures.

†This was a young woman with temporal lobe epilepsy (M.G.). She was thought at the time of operation to have a deep temporal neoplasm and a subtemporal decompression was carried out. She has had no attacks in the 17 years since operation and shown no evidence of tumour growth.

temporal lobe epilepsy and who were treated by partial temporal lobectomy in this period.*

For the sake of brevity we will divide them into two groups: (1) Successful (patients who have had no more than three attacks in the first three years after operation and most of them no attacks). (2) The rest we may call unsuccessful, although many in this group were much benefited.

Using this definition, 32 patients or 47% had successful results and 35 unsuccessful. The follow-up period varies from one to more than seven years; in 32 of the patients it is more than three years.

The results show that recurrence of seizures is most likely in the first six months after operation, and that for this group none recurred after three years. Of course, we realize that recurrence is possible at any time. Considering only the patients followed up for over three years, 16 or exactly one-half had a successful result.

Eighteen patients had an upper quadrantic field defect after operation, but in none was it disabling. Temporary aphasia occurred in 11 patients but persisted in only one. Twenty patients complained of memory defect, but this was severe and disabling only in one.

The pathological lesions discovered at operation were: atrophic lesions with or without cysts in 38 cases, microgyri in six, meningocerebral cicatrices in four, angioma in one, hæmangioma calcificans in four. In 13 cases the only abnormality noted was increased consistency of the gray matter deep in the temporal lobe.

I. Comparison with Earlier Series and Factors that Influence Results

We may now return to a reconsideration of Table VII which shows the results of cortical excision in the 203 patients of this series. It will be seen that there are 57 patients with perfect results (28% of the total), and this should be compared with the earlier figures of 24 (21%) of 115 patients (Penfield and Erickson) and 15 (25%) of 59 patients (Penfield and Steelman). There appears to be a slight improvement in the number of perfect results with each successive series. In regard to the other follow-up groups a more severe standard of selection is set in the present series as compared with the earlier ones.

In this present series, 34 patients achieved a nearly perfect result (group 3), only 17% of the total as compared with 25 patients (22%) of the first report and 18 (30.5%) of the second. At first glance this would appear to be less satisfactory in the latest series, but the classifications of group 3 in the three series are not strictly comparable. In the first study, a few patients with one or two seizures a year were included in group 3 and in the second a 75% improvement was classed as group 3. This might include patients who were having one or two attacks a year and who would in this series be classed in group 2, and also include those who had been free of attacks for three, four or five years up to the time of follow-up, but who might have had a sufficient number of seizures in the first year or two after operation to exclude them from group 3 by the standards adopted for the present analysis.

In this series, 37 patients (18%) derived slight benefit from operation, and this can be contrasted with 11 (9.6%) of the first and 7 (12%) of the second series. Those patients who were not benefited by operation or were made worse (very few of the latter) numbered 35 (17%), whereas in earlier operations 25 (22%) and 11 (18.6%) had this unsatisfactory outcome.

Group 2 patients in this present series are even less comparable with those of the earlier reports. Forty patients have had a satisfactory result after operation, placing them in group 2—that is, 20%. If one could guarantee a group 2 result, as graded in this study, to every patient, cortical excision would be considered a useful procedure.

The higher incidence of perfect results in the last series suggests that we are deriving benefit from longer experience, although the incidence of failures remains about the same. The chief changes are the ever-greater reliance placed upon electrocorticography during operation and the great increase in temporal lobe cases.

The percentage of patients with successful or satisfactory results in this series has remained just below 70% over the years, despite the wide extension of operation year by year to cases previously considered unsuitable for surgery. Complete freedom from attacks (see Table X below) applies to almost half of the patients when considered year by year.

Thus there are patients, in groups 3 and 2, who have had no attacks for a certain period of years up to the time of their follow-up. Fifteen patients in group 3 have had no seizures in the last one to three years and three patients included in group 2 have had no attacks in that period. Phenobarbital was the only medication used by 11 of these patients, three are taking anticonvulsants other than phenobarbital and

*Some of these patients were reported by Penfield and Flanigin (1950) but others are added and the follow-up period is extended. There were 19 other patients who had temporal removals in addition to excision elsewhere. They are excluded. Since 1950 the proportion of temporal lobe cases has steadily increased so that they now compose well over 50% of the total patients who come to operation.

three are taking no drugs. In one case, the medication is unknown.

In addition to the above, seven patients in group 3 and one patient in group 2 have had no seizures for more than three years up to the time of follow-up. Five of these are taking no medication, one phenobarbital only and the other two anticonvulsants other than phenobarbital.

In females the rate of success was higher than in males, and this was especially so in those patients with lesions due to birth or postnatal trauma.

At follow-up, 48% of patients with epilepsy following a birth lesion were classed in group 3 or 4, but only 39% of those with post-traumatic seizures. The remaining etiological subdivisions are too small for comparisons.

Those patients whose seizures had been present for less than two years before operation had a better success rate—54%—than the remainder—only 44%. However, it is interesting that of the patients (24 in number) who had their seizures for over 20 years, 46% could be said to have had a successful result.

Well-localized electroencephalographic foci, with or without transmission of the abnormal wave forms to the opposite side, and indicative of a localized epileptogenic lesion which could be excised, promised a better prognosis—47% in groups 3 and 4—than were bilateral abnormal discharges with uncertain localization—only 33% success.

Excision of the frontal area seemed to be more favourable—54% success—than other excisions, the worst being perisylvian and parietal with only 27% of patients placed in successful groups after excisions in these regions.

Early postoperative seizures are of bad prognostic import, whether the seizures be similar to the preoperative attacks or not. The electroencephalographic recording three or more weeks after operation is a comparatively good guide to the ultimate result.

J. The Patient's Assessment

The patient's own opinion of the operation is worth recording briefly. All patients in group 4 were satisfied, but three in group 3 believed themselves worse off because of some physical defect following operation. Even in group 0, there were six grateful patients. Altogether there were 40 patients who thought themselves worse after operation, out of a total of 150 who expressed their opinion in this regard.

There were 125 patients, each of whom expressed his assessment of the result of operation in percentages at the time of this survey. This personal assessment is shown in Table VIII. Many elements are, of course, included in this voluntary expression of opinion—disabilities of various types, emotional sequelæ and, most important, freedom from attacks.

TABLE VIII.

PATIENT'S ASSESSMENT OF RESULT

"100% cured"	39	31%
"Over 75% improved"	25	20%
"Over 50% improved"	37	30%
"Over 25% improved"	1	1%
Not improved	23	18%
Total who gave opinion	125	

K. Multiple Operations

When the result of cortical excision is not satisfactory a second procedure may be carried out in selected cases to extend the removal in one direction or another. Occasionally, a failure may thus be converted into a brilliant success.

During the years of our analysis, 27 of the patients adequately followed up were operated upon twice.* Nine of them were converted from failures to group 4 successes and two became group 3 successes. Thus 41% of the 27 patients reached the successful groups and another 19% were in the satisfactory group (2). Within this series a second operation proved to be worthwhile in 60% of the patients thus treated.

The epileptic patient should be interested in the control of attacks throughout his life span. In our experience there is apt to be little change in the result of operation after a three to five-year postoperative period, although there may be steady improvement far beyond that period, as we have learned from our early cases.

In Table IX the results for the 51 patients in this series, whose follow-up is longer than five years, are set out according to our method of group tabulation. It is seen that 41% of the patients are in successful groups five to seven years after operation. But this actually gives an erroneous impression because, according to our

*This does not include the operative deaths. One such patient had had 4 operations. Two had 2 operations. There was another patient who had 2 operations but is excluded because he could not be followed up.

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TABLE IX.

RESULTS IN 51 PATIENTS FOLLOWED UP FOR 5 TO 7 YEARS

Group 4.	Perfect result.....	12	(23%)	Success 41%
Group 3.	Nearly perfect result	9	(18%)	
Group 2.	Satisfactory result..	18	(35%)	Good 35%
Group 1.	Slight improvement	6	(12%)	Failure 24%
Group 0.	No improvement...	6	(12%)	

method of recording, a patient who does badly for a few months can never get into the successful groups, even if his attacks should stop completely.

When the whole group is considered it is recognized that some patients drop out of the success groups because of some recurrence, perhaps transient, but others become seizure-free in later years, making for a compensating balance.

When each of the years that follow operative excision is taken by itself without regard to other years, it appears that there is a reasonably steady quota of patients who are completely seizure-free. Table X shows that this quota varies between 40% in the first postoperative year and 48% in the sixth.

TABLE X.

FREEDOM FROM SEIZURES YEAR BY YEAR

Year after excision	*Total patients followed up*	*Complete freedom from attacks*
1st	203	80 — 40%
2nd	173	77 — 45%
3rd	124	50 — 41%
4th	86	35 — 41%
5th	51	21 — 42%
6th	25	12 — 48%
7th	11	5 — 45%

L. EFFECTS OF EXCISION ON FUNCTION AND EFFICIENCY

It is not enough to know whether a radical surgical procedure has stopped attacks or not. We must know its effect upon the patient's ability to work, to hold a job, to study; the effect on physical and mental function, the effect on behaviour and on the happiness of patient and friends. When all the features of his life are considered, it still remains for the physician to ask the final question: In the opinion of the patient and of those who love him, was the operation a success or a failure? This is answered in part by Table VIII, but we may examine the question in more detail.

ECONOMIC RESULTS

An economic analysis is not easy. Before operation 79 out of the 203 patients were fully occupied in spite of seizures. Not infrequently it was the threat of loss of employment that brought the patient to seek operation. Five of them are not now employed and two are in mental or epileptic homes. On the other hand, 51 not previously employed are now on a satisfactory full-time work or educational schedule. There are 38 patients out of work but possibly employable when last heard from.

PHYSICAL EFFECTS

The child who is hemiplegic, epileptic and progressively more and more retarded mentally seems to have a great deal to gain and little to lose. If the attacks are stopped, mental progress and behaviour improved and spasticity of the paralyzed extremity lessened, then it is obvious that everything is achieved that could be hoped for. But if only one of these things is accomplished, if the attacks stop and the child does better in school, the parents may call the procedure a splendid success although the paralysis is unaltered.

If a man who is physically and mentally normal has his seizures stopped but forfeits the use of an arm so that he cannot work or develops a serious defect in memory, he may consider the procedure a failure. On the other hand, there are other bodily disabilities he would gladly accept to escape from the curse of seizures, for example a hemianopic defect or a cortical somatic sensory loss.

Clinical cases are personal as much as they are scientific problems, and the clinician must often make the best compromise with perfection that he can. He must understand the patient and his hopes before he presumes to decide on treatment. But this is, after all, the secret of the art in the practice of medicine.

Frequency of attacks after operation is obviously not the only measure of success although it is usually the most important.

Hemiplegia or hemiparesis, aphasia and memory loss are the serious defects that a neurosurgeon fears as a sequel to cortical removal or the end result of some untoward complication of operation. Cortical sensory loss alone or a de-

fect in the homonymous visual field is usually considered an unimportant deterrent when there is a reasonable chance of stopping seizures.

Hemiplegia or hemiparesis as an untoward complication may result from manipulation of the middle cerebral vessels, especially when scarred cortex is removed above the fissure of Sylvius as well as below it. It may be difficult to decide whether this has produced arterial thrombosis or simple spasm. There is also some danger of trauma to the internal capsule during the process of removal of the cortex of the insula or the cortex deep within the fissure of Sylvius. Then there is, of course, always the danger of hemiplegia that may be caused by postoperative bleeding unless reopening is carried out in time. But this is a complication that all neurosurgeons must guard against after every craniotomy.

Out of 159 patients who had normal or almost normal motor and sensory function before operation 15 were found to have somatic motor or sensory abnormalities at the time of follow-up assessment. Six of these abnormalities were considered to be severe. Two were hemiplegias. In all, 26 patients had some degree of motor or sensory disability not present before operation, often minimal and usually anticipated. In all but five the excision was carried out in the central area.

On the other hand, in our analysis of these cases there would seem to be eight patients whose paralysis or weakness was improved by operation. Six were converted from a severe to a moderate defect and two from a moderate defect to almost normal.

Among these eight patients who claimed improvement in somatic motor function, four had removals of part of the central or Rolandic cortex, two had temporal lobectomies, one had intermediate frontal (supplementary motor) removal and one had extensive removal of central, parietal and temporal cortex.

This improvement seems, at first sight, difficult to understand. In some cases there was "paradoxical improvement" in the existing hemiplegia (as described by Welch and Penfield, 1950) resulting from reduction in spasticity when a useless precentral gyrus was removed. In others it seems possible that there was great weakness secondary to a bout of seizures before operation and that this weakness may have cleared up or improved as the result of cessation of seizures.

CHANGES IN VISUAL FIELDS

Thirty-two patients developed a visual field defect as a result of operation, or a pre-existing defect was increased by operation. As one would expect, these followed excisions in the posterior temporal and occipital regions. Temporary lobectomy frequently gave an upper quadrantic defect, but the patient did not report this as a disability. The only complete hemianopic defects were produced by occipital lobectomy.

Temporal lobectomy was followed by a field defect (upper quadrant anopia) in 18 patients, and it is of interest to correlate the extent of lobe removal with the visual defect. The surgeon usually measured the length of the amputation from the anterior pole of the lobe, so that figures are available. The figures must be taken with some reservation, of course, as it is the injury to the deeper optic radiation which is important and not the extent of superficial cortical excision.

Excision of the anterior 4 cm. of the lobe produced a defect in only one patient out of 14. When the removal amounted to 5 cm. or a little more, 5 out of 20 had postoperative field defects. Four out of 12 had defects if 6 to 7 cm. were removed, and 3 out of 8 when the ablation extended 7 to 8 cm. All three of those with more than 8 cm. of the temporal lobe removed had a field defect. In two cases the extent of lobectomy is not known.

It must be admitted that completely accurate visual field examinations were not carried out on all patients of this series, but more detailed recent studies have provided interesting results which will be published eventually.

ASPHASIA

After cortical excision carried out in the dominant hemisphere, aphasia made its appearance frequently during the postoperative period, at the usual time for neuroparalytic œdema, about the 3rd to the 5th day after operation. It cleared up about the 10th to the 14th day. Such transient effects will not be referred to again.

In all cases, excisions of this type were carried out under local anæsthesia so that the patient could continue to talk. When no aphasia appeared while the patient was on the operating

table, its subsequent appearance proved to be of little moment.*

Aphasia was present when the patient was discharged from hospital (14th to 21st day) in the case of 15 patients, and it persisted as a disability in only three patients.

It is possible that the remaining 12 patients discharged with a definite aphasia would show some changes at the present time if they were available for careful examination, but they themselves and their relatives do not recognize any difficulty in speech, nor is there any complaint of defect in reading or writing.

Two of the patients were lefthanded but all had speech localization in the left hemisphere. Eleven of the 15 speech defects were produced by temporal removals as follows: one after a 4 cm. removal, two after 4 to 5 cm. removal, seven after more than 5 cm. removal. In the 11th case the extent of removal was not accurately recorded. In three patients the excision was in the left lower intermediate frontal region, and the remaining excision was in the left lower centro-parietal junction.

INTELLIGENCE

No special study of the mental state before and after operation was made. But some idea of mental change may be had from the clinical notes. Psychological and psychiatric studies are in progress now and will be published. Memory loss was discussed above under the heading of Partial Temporal Lobectomy.

It would appear that, in all, 24 patients have deteriorated since cortical excision. None of them was in the success group 4 and only three were in the success group 3. Thus the intellectual deterioration occurred largely in patients who were not relieved of their seizures and one may surmise that it was associated with the continuing seizure process.

M. SUMMARY NOTE

This is a report of the results of the surgical treatment of 234 patients who had focal cerebral seizures together with a discussion of the reasons for success and failure. This series includes all such patients operated upon in the six-year period that closed in 1951, excluding cases of neoplasm. When added to the two preceding follow-up studies, 470 such cases may now be reconsidered.

Before operation none of these patients had been able to lead happy, efficient lives, even with the help of the best regimens of conservative therapy. The procedure of cortical excision, which was recommended in the first place as a counsel of despair, has come to be applied to patients in a rapidly expanding field. Mechanisms of cause and techniques of study and treatment are described and the chances of success evaluated.

For three-quarters of the patients the operation proves to be a real benefit, and half of the patients continue to be completely free of attacks year by year, at least as far as our analysis goes, into the seventh postoperative year.

Epileptogenic cortex is nociferous or harmful cortex because of its general influence on brain function. Successful removal, when this is possible, may result in better intellectual performance and more normal behaviour as well as in freedom from seizures. There are the dangers of operation and the risks of functional deficits to be considered. But in spite of this the treatment of focal epilepsy by surgery is now established as a legitimate form of therapy.

Success demands special training, and the neurosurgeon must have clinical and scientific co-operation in a well-equipped hospital. But there is no field in medicine where the rewards are greater in terms of human happiness and efficiency.

*A method of speech localization exploration has been used which will be reported in detail with our associate Dr. Lamar Roberts. A stimulating electrode is applied to the cortex to produce local interference with function. If aphasia appears during stimulation, and if there is no evidence of spreading after-discharge when the electrode is removed, the convolution stimulated is considered essential to speech and is carefully avoided during excision unless there is other strong evidence in favour of its removal.

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RÉSUMÉ

Le but de cette communication est d'évaluer le traitement de l'épilepsie par les excisions corticales. La nature même de l'irritation qui rend la matière grise épileptogène est encore inconnue. D'après Hughlings Jackson, chaque crise épileptique commence par une décharge neuronale de la formation grise. Si, théoriquement, cette aire est enlevée, les crises devraient cesser. Cependant, la difficulté consiste à identifier et à enlever tout le cortex épileptogène, mais non plus. Le foyer est l'endroit où les potentiels électriques sont les plus élevés entre les attaques; cependant, le cortex epilepto-

gène peut être beaucoup plus vaste que ces indications nous permettraient de le soupçonner. Ce foyer peut être localisé cliniquement d'après la manifestation initiale de la crise, ou à l'aide de l'électro-encéphalographie. A l'opération, on peut aussi quelquefois reconnaître le cortex anormal à la simple inspection. La stimulation électrique du cerveau même peut reproduire la manifestation initiale de la crise. Les lésions pathologiques peuvent être une tumeur, un kyste, une zone de destruction résultant d'une hémorragie, d'une thrombose, ou d'un traumatisme produit à la naissance, lésant le cortex adjacent. L'électro-corticographie est la baguette de coudrier du neuro-chirurgien à la découverte de zones épileptogènes. L'entité pathologique la plus fréquemment vue est la "sclérose par l'incisure" de l'hippocampe et de l'uncus (vide infra). La dureté du cortex épileptogène proviendrait du changement des astrocytes protoplasmiques en astrocytes fibreux et de la prolifération de ces cellules. Une particularité commune à toutes ces lésions semble être une insuffisance de la circulation, minime mais persistante. L'importance de ce désordre n'est pas encore bien établie. Un trouble du métabolisme de l'acétylcholine par le tissu épileptogène a été incriminé par Elliott, et paraît relié à certains degrés d'anoxie.

L'auteur présente une série de 234 malades ayant subi une craniotomie pour le traitement de l'épilepsie, entre 1945 et 1950. Aucun d'entre eux ne présentait de tumeur intra-cranienne. Tous les malades ont été suivis pendant au moins un an; certains d'entre eux l'ont été pendant 7 ans. Cent quatre-vingt-dix d'entre eux répondirent à un questionnaire écrit; bon nombre de leurs proches parents y ajoutèrent leurs propres commentaires.

Les résultats montrèrent une distribution pour les hommes de 65%, et de 35% pour les femmes. Cet excédent ne s'applique pas seulement à l'épilepsie post-traumatique mais bien aussi aux accidents de la naissance et à l'anoxie néo-natale. Dans 4.4% des cas, il fut possible de trouver des antécédents familiaux de désordres convulsifs. Les tendances héréditaires peuvent donc être laissées de côté. Sous le titre d'accidents de la naissance furent réunis les traumatismes par instrumentation, les hémorragies intra-craniennes, l'anoxie cérébrale et l'herniation du cortex temporal par l'ouverture de la tente. La "selérose par l'incisure" est décrite par l'auteur comme étant la lésion produite par la compression temporaire des artères qui franchissent l'échancrure de la tente pour irriguer l'uncus, l'hippocampe et le cortex adjacent, par l'engagement des circonvolutions temporales vers la fosse postérieure. Les convulsions produites dans un état fébrile ne jettent aucune lumière sur la nature de la lésion cérébrale puisque la fièvre ne peut servir qu'à démasquer la présence d'une lésion atrophique, préalablement occulte. Les crises du lobe temporal commencement le plus souvent entre les âges de 5 et 20 ans. Les premiers-nés se retrouvent plus souvent dans les malades souffrant d'épilepsie focale causée par un accident de la naissance que le reste de la population en général. Dans les cas douteux de lésion traumatique post-natale, la présence d'hémiparésie, de déficit sensoriel ou d'aphasie datant du temps de la blessure, fut prise comme preuve de lesions localisées du cerveau. Le tableau no. 4 montre que l'intervalle entre le traumatisme post-natal et le début des crises habituelles est d'environ un an après les blessures pénétrantes de la tête et d'un peu moins d'un an pour les blessures non-pénétrantes. Dans les lésions intra-craniennes suppurées, les crises apparurent entre 2 et 13 ans après la phase aiguë de l'infection.

Les sept angiomes de la série furent tous excisés, ainsi que les circonvolutions adjacentes, sauf un de la région centrale droite dont l'enlèvement aurait pu causer une grave hémiplégie. La dégénérescence des hémangiomes calcifiants est prononcée et leur vascularité est beaucoup diminuée. Les 4 cas de cette série se trouvaient dans le lobe temporal.

Sous l'en-tête "Divers" furent réunies 18 lésions disparates telles que: l'ischémie post-natale, l'encéphalite du plomb, la malaria cérébrale, la sclérose tubéreuse de Bourneville, l'urémie, les encéphalites (probablement à virus), la maladie hémorragique du nouveau-né, les possibilités de tumeur cérébrale, les méningites, et l'artériosclérose.

L'auteur désire attirer l'attention sur le délai considérable dans le début des crises répétées causées par les lésions temporales datant de la naissance. Chez les 73 malades dont les crises commencèrent entre les âges de 10 et 20 ans, la cause se retrouve indifféremment entre le traumatisme de la naissance, le traumatisme post-natal, etc. Pour servir aux fins de cette étude, l'attaque d'épilepsie devait comprendre au moins une des conditions suivantes: mouvements convulsifs, perte de connaissance ou perte de mémoire. Aucun des malades ne reçut de médication anti-convulsive après l'opération, sauf du phéno-barbital. Ceux qui durent recourir à la phénytoïne (dilantin) furent considérés comme des échecs chirurgicaux. Des 203 cas ayant subi une craniotomie et qui purent être suivis par après, 57 n'eurent plus jamais d'attaque et 34 n'eurent que trois attaques ou moins avant que les crises ne disparaissent. L'intervention peut donc être considérée comme un succès dans 45% des cas. Des résultats satisfaisants furent obtenus dans un autre 20%. Des 68 malades ayant subi une lobectomie temporale partielle, 47% bénéficièrent d'un bon résultat. La comparaison avec des séries semblables publiées antérieurement montre une proportion croissante de résultats excellents que l'on peut attribuer à l'expérience acquise. Deux facteurs nouveaux sont cependant apparus: une plus grande fréquence des lésions du lobe temporal et une plus grande importance attachée à l'électro-corticographie. Les bons résultats furent plus nombreux chez les femmes que les hommes. Cette différence a été particulièrement marquée dans les cas de lésions relevant de traumatismes natals et post-natals. La reprise des attaques tôt après l'opération est de mauvais augure. Trois semaines ou plus après l'intervention, l'électro-encéphalographie offre un indice assez satisfaisant des résultats à venir. Lorsque ceux-ci ne sont pas satisfaisants, une deuxième intervention peut être pratiquée dans des cas choisis. Un certain nombre d'échecs devinrent ainsi de brillants succès et, dans 60% des cas, cette deuxième opération s'avéra utile.

Au point de vue économique, 51 malades sont maintenant à l'ouvrage et 38 autres sont en mesure de l'être. De 159 malades ayant une fonction motrice ou sensorielle normale ou quasi-normale avant l'opération, 15 subirent une perte motrice ou sensorielle, 6 d'entre eux à un degré marqué. Par contre, la paralysie ou la faiblesse pré-opératoire de 8 malades fut améliorée par l'intervention. Les défectuosités du champ visuel, résultant de l'opération, n'ont pas semblé nuire aux sujets qui en furent atteints. Une aphasie passagère s'est souvent manifestée vers le 3e ou 5e jour après une intervention intéressant l'hémisphère dominant; deux semaines plus tard, elle était disparue dans la plupart des cas. Quinze malades présentaient encore de l'aphasie a leur départ de l'hôpital; elle ne persista que chez trois d'entre eux. Une détérioration mentale s'est produite chez 24 malades ayant obtenu peu ou pas d'amélioration de leur opération. Il est possible que cette détérioration soit en rapport avec la persistance des attaques. M.R.D.

MEDICAL PRACTICE IN MANITOBA

The Students' Guide number of the *Lancet* (August 27) contains a nice piece of Canadian enterprise in the form of an article by Drs. McLandress, Martin and Briggs of Winnipeg, describing medical practice in Canada with special reference to pædiatrics in Manitoba.

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A MUSCULAR DYSTROPHY CLINIC

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THE LAST FOUR YEARS have seen rapid growth of the Muscular Dystrophy Associations of America, and last fall the Muscular Dystrophy Association of Canada received a charter as a non-profit health organization. What have these bodies achieved to demand recognition?

MUSCULAR DYSTROPHY ASSOCIATION OF AMERICA

The American body has established a reputation with the public and the medical profession as an efficient, far-sighted group that has within three years attained a position in which during 1953 it was able to collect nearly $4,000,000. Of the net income, 75% is always earmarked for research, and over $1,000,000 was already allotted during 1954. This and earlier annual collections currently cover the cost of 64 research projects at universities in the U.S.A. and one in France. Since only $44,000,000 is available from all sources for medical research in American medical schools, the sum of well over $2,000,000 for muscular dystrophy research constitutes a sizable proportion of the total. Such a response from the public is impressive, but more important is the question of how this money is being used. At the third medical convention of M.D.A.A. in October 1954 in New York, 33 medical papers[8] were presented. Basic physiology, electron microscopy and biochemistry were much in evidence, and it is likely from the wide studies undertaken that problems in other related disorders may also be solved in the search for the cause of muscular dystrophy. The wise use of this money for research has been guided by a distinguished medical advisory board under the chairmanship of Dr. A. T. Milhorat, Associate Professor, Cornell University Medical College, who has himself made notable contributions to the knowledge of this disease.

M.D.A. OF CANADA

The newly established Muscular Dystrophy Association of Canada took part in a North American appeal for funds in November 1954. Incomplete figures indicate that a sum approaching $300,000 was collected in Canada. As in the United States, 75% of these funds is earmarked for research. It is hoped to carry out a co-ordinated programme at Canadian universities in the near future. Research projects already approved include two at the Hospital for Sick Children, Toronto, one at the University of Toronto, one at the Montreal Neurological Institute, one at the University of British Columbia, Vancouver, and one at the University of Saskatchewan, Saskatoon. By liaison with the Muscular Dystrophy Association of America, duplication of effort is being avoided.

M.D.A. CLINICS

A number of special clinics for muscular dystrophy cases have been established in the United States. While the need for basic research is clear, the utility of special clinics for what is still an incurable disease might well be questioned. The best answer is in the results reported by existing clinics.

RESULTS

1. *Physical.*—As in poliomyelitis, contractures are often a problem, particularly in the calf muscles. Often it is this which finally makes the patient give up walking. Judicious stretching by a physiotherapist—leaving just enough equinus to aid knee extension with a weak quadriceps—is often of value. It has also been shown[5] that a course of exercises—resisted where indicated—carried out three times a day for three months will cause appreciable increase of power in the residual muscles. The improvement, though slight, may be worth while in some cases. It is also clear that before any trial of a new drug is carried out in a muscular dystrophy patient, the muscles to be tested must first be brought up to their fullest capacity by such physical means.

In some cases splints will keep a patient ambulant, but the splints must be exceedingly light in weight. Adjustments of shoes may correct increasing varus or valgus. In some cases a back brace fixed to a wheel chair will enable a patient to sit up rather than take to bed in the later stages.

Arm slings attached to a wheel chair or bed, or hinged horizontal arm rests attached to a

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wheelchair or pelvic brace are valuable when arms are weak. These and many other devices[a-e] in use for poliomyelitis cases may be suitable also for muscular dystrophy cases. The emergency use of a respirator when an advanced case of muscular dystrophy develops a respiratory infection is just as life-saving and urgent a measure as in poliomyelitis patients who have been left with a respiratory defect. In both cases, mere treatment of the infection without recognition of the basic need for increased aeration is likely to be lethal. In late cases of muscular dystrophy life may be prolonged for months or years by regular use of a respirator; a mobile chest-piece type (Huxley, Emerson) or the new model of wheel-chair respirator[1] will enable the patient to get outside the house and visit friends or go to movies for five hours before the battery requires recharging.

Surgical procedures may occasionally be worth while, but any procedure which requires inactivity for more than a very short period should scarcely ever be considered, as these patients may never regain their balance and ability to walk if disuse atrophy is added to the existing severe wasting. Most of them walk precariously with a gluteal gait and a marked lumbar lordosis, and the adult in particular may continue many years with just enough strength to navigate. Some patients become obese and may be helped considerably by reducing, while others must be dissuaded from starving themselves to the point of emaciation to reduce the load they have to carry.

2. *Psychological and social.*—The benefit to these patients—as with many other handicapped people—of meeting others who are cheerfully carrying on under difficulties is well worth the effort made by the patients, their relatives and friends in organizing a local chapter, and by doctors in running a clinic. Of equal value is the interest stirred up in the community. This can lead to job placement and help with transportation, quite apart from the mutual aid plans such as baby-sitting duties, with which one family can help another. The dramatic change in temperament from a shy, retiring, self-centred frustrated personality to one with a cheerful, outgoing, determined and hopeful outlook has occurred often enough in the handicapped to justify any amount of work done for them.

3. *Eugenic.*—In some dystrophy families of my acquaintance, a member has gone ahead with procreation without warning his spouse of the risk of passing on the disease. In others, members have remained childless for years on the mistaken supposition that they might be carriers. To both of these attitudes, the only possible answer is education of both patients and their blood relatives. This involves careful family history and examination of all concerned, to make sure that the apparently unaffected do not have the disease in a latent form which would render them just as liable to pass it on as if they were advanced cases.

Probably the finest contribution to heredity studies of muscular dystrophy was made and is still being made by Tyler.[1-4] He has greatly simplified the classification of muscular dystrophy by reducing it to: (1) the childhood type; (2) the facio-scapulo-humeral type; (3) dystrophia myotonica.

This division enables one to forecast with considerable accuracy the likelihood that any given member of a muscular dystrophy family may convey the disease to his or her children, since the inheritance is sex-linked recessive in the first, and dominant in the last two categories. The varieties breed true to type, and Tyler found only four cases out of several hundred which could not reasonably be placed in one of these groups. The childhood type usually affects males, is passed on by unaffected females, and almost always starts between the ages of one and eight (usually two to five), while the facio-scapulo-humeral type can affect either sex and very rarely begins before the age of seven (usually 10-17). While the childhood type is fatal in five out of six cases before the age of 21, the facio-scapulo-humeral type is commonly compatible with a normal life span, though it is an increasing handicap and may eventually cause complete invalidism. The younger the age at which either type 1 or 2 starts, the more rapid a course it tends to run. For further details, Tyler's papers should be consulted. With Tyler's method of classification one feels on surer ground than with

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(a) Georgia Warm Springs Foundation, Warm Springs, Georgia. Extensive photographic records of special appliances available on request.
(b) Self-Help Devices for Rehabilitation, obtainable from New York University—Bellevue Medical Center, Institute for Physical Medicine and Rehabilitation, 400 East 34th Street, New York 16, N.Y., U.S.A.
(c) Robin Aids, 2015 Broadway, Vallejo, California.
(d) Lionel Electro-Mechanical Hand. The Lionel Corporation, 28 Sager Place, Irvington, New Jersey, U.S.A.
(e) Saber Arm, North American Aviation Inc., International Airport, Los Angeles 45, California, U.S.A.
(f) Wheelchair Respirator Corporation, 1424 K Street N.W., Washington 5, D.C.

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the previous use of the term pseudo-hypertrophic muscular dystrophy, since this type of muscular swelling can occur in both types 1 and 2. Age at onset is a more reliable guide to the variety of the disease. It should also be noted that in about one-third of cases of the childhood type the disease arises as a new mutation, whereas a family history can almost always be obtained in the facio-scapulo-humeral type.

It is good to be able to reassure an unaffected male member of a childhood dystrophy family that there is little risk of his passing on the disease. Similar information can be given to an unaffected member of the facio-scapulo-humeral type family, once the age of 25 has been attained, provided an expert examination is negative and reveals none of the *formes frustes* of the disease — specifically, large calves, small sternomastoids, abnormal movements of orbicularis palpebrarum, and absent tendon reflexes. Inability to raise the arms above the head may be the only sign of facio-scapulo-humeral dystrophy for many years. Atrophy of brachioradialis, trapezius and lower fibres of pectoralis major may also be present early. A biopsy of an involved muscle in a doubtful case may settle the matter. It must be added that Tyler's views on heredity and classification of this disease, while very well supported by detailed clinical studies, have not yet had time to become generally accepted, and other prominent authors[6] still stress the apparent overlap between different types of muscular dystrophy, the possibility of the disease developing in later life, and of its being transmitted by any member of a muscular dystrophy family, whether affected or not. It is possible that there may be differences in the hereditary pattern in different kindreds, and it is not safe to generalize at the present time. *Responsibility for giving eugenic advice in these cases should be shared with an experienced specialist.*

4. *Economic.*—Many of the patients waste a small fortune on useless treatment by drugs, vitamins, diets, electrical gadgets and manipulations. The clinic can obviate this and make sure that their money is conserved for more useful purposes.

5. *Educational.*—Much of the knowledge is new, and cases are rarely seen. Hence doctors, as well as patients and their relatives, gain much if the cases are gathered together into a special clinic. Interns and students can also be taught at such a clinic and cases of doubtful diagnosis can be referred and discussed.

6. *Research.*—Out of the extensive studies of this disease in research laboratories—or perhaps, like penicillin, as a gift from the sky—there may one day be found an effective cure or prophylactic for muscular dystrophy. When that day comes there will be need for well-organized clinics where the new remedy can be tested under controlled conditions. This will obviate the fiasco of a well-publicized remedy which eventually proves useless. Dr. Milhorat,[7] in a recent work, lists over 80 such drugs and other therapeutic agents which were heralded with enthusiasm, but proved quite unreliable in practice.

HAMILTON CLINIC

In the summer of 1954 the Hamilton (Ontario) chapter of the M.D.A. of Canada suggested that a clinic be organized in Hamilton. Space was granted free of charge at the Hamilton General Hospital, and the first clinic was held on December 6, 1954. In view of the obscure nature of muscular dystrophy it seemed that a more intensive individual case study was needed than the average out-patient clinic affords, and that experience should be drawn from several specialities. These desiderata were fulfilled by getting together the following team, each member giving his time and services free of charge: a general and an orthopædic surgeon, an internist, a neurologist, a pædiatrician, a psychiatrist and a general practitioner.

A list of over 50 muscular dystrophy cases in the Hamilton vicinity was provided by the secretary of the chapter. Only two or three cases are seen at each two-hour weekly clinic, and each is presented by one of the team who has already prepared a report, having examined the patient and studied his family and social history and a muscle chart completed by a physiotherapist. The team makes joint recommendations, and the chairman has authority to arrange for any requisite treatment, the cost being met by the funds of the chapter when necessary. Thirty-seven cases were studied in the first six months, almost always with some useful outcome.

SUMMARY

1. A brief history is given of the Muscular Dystrophy Associations of America and Canada, stressing their contribution to research.

2. The rationale for special muscular dystrophy clinics is discussed as regards the patient, his relatives, and the medical profession. These clinics make a practical contribution in a way that is physical, psycho-social, eugenic, economic, and educational and pave the way for drug trials or other clinical research.

3. The starting in Hamilton, Ontario, of the first clinic for muscular dystrophy cases in Canada is recorded. Intensive study of over 50 cases by a group of specialists as a public service is in progress.

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Since this article was written a second Muscular Dystrophy Clinic has been opened at the Hospital for Sick Children in Toronto.

ANTRENYL*—A CLINICAL EVALUATION

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THE SEARCH for an efficient method of controlling hyperacidity and hypersecretion, as well as disturbances of gastrointestinal motility, has perhaps, at least partially, been motivated by the dissatisfaction of the clinician with atropine and belladonna administered by the oral route, as exemplified by Bastedo's publication of 1936.[1] Considerable impetus has been added to this search by the publications of Grimson and his associates[2, 3] on Banthine (methantheline bromide). While the initial enthusiastic acceptance of this product was perhaps unduly optimistic, the swing of the pendulum has brought with it a frequently expressed opinion that the efficiency of atropine and belladonna may have been unjustly slighted. To quote from a recent publication[4]: "We have no evidence to indicate that Antrenyl will do anything, physiologically or therapeutically, which atropine in adequate dosage will not do." Later in the same publication, however, the authors state: "We have not observed any harmful effects of Antrenyl in the human in periods up to eight months in doses up to 100 mg. per day throughout that time." This is five times the average therapeutic dose, and it is doubtful whether atropine or belladonna employed in equivalent quantities, and for a similar period of time, would prove as innocuous. A substantial number of publications stress that the desired results can be obtained only with very high or toxic doses of these drugs.[1, 5-10]

While there is ample evidence that the effect of atropine and belladonna is considerably enhanced by parenteral administration,[10-13] Levin and his associates[12] point out that this enhancement is frequently associated with toxic symptoms; nor does this method lend itself to the prolonged administration required in the treatment of peptic ulcer. A great service would be rendered to both patient and physician if the relative merits and shortcomings of the old and the new anticholinergics could be clearly established.

Some of the more recent literature tends to throw light on this subject. While there is more or less agreement that the antisecretory effects of orally administered newer anticholinergics are variable and more or less unpredictable,[14] Broicher[15] maintains that the oral administration of Antrenyl (oxyphenonium bromide) is considerably more effective than that of atropine. The effect of Antrenyl on motility and spasticity appears to be more predictable and more readily attained.[14-17] Demling and his associates[16] found Antrenyl to be superior to atropine and to five of the newer anticholinergics, in respect to both efficacious spasmolysis and the duration of the effect. Ruffin and his associates[14] refer to the pronounced alterations in motility attained with parenteral administration of anticholinergic

*Supplied by Ciba Company Ltd., Montreal.
†From the Department of Medicine, Jewish General Hospital, Montreal.

TABLE I.

Case No.	*Disease*	*Duration*	*Therapy prior to Antrenyl*	*Results with Antrenyl*	*Reactions*
5 D.R.	Duodenal ulcer	Years	Alkalis, antacids (F), Banthine (P)	Good	Dribbling, slight
9 P.M.	"	2	Sedatives (P), antacids (P), Banthine (P)	"	None
80 J.N.	"	30	Sedatives (P), antacids (P), belladonna (F)	Excellent	"
12 E.N.	"	7	Sedatives (P), antacids (P), Banthine (P)	"	"
13 A.O.	"	2	Banthine (P)	"	Slight constipation
23 S.T.	"		None	"	None
26 G.Z.	"		Antacids (P), Triloid (P and M)	Partial	Dulling senses
28 M.M.	"		Sedatives, belladonna (F)	Excellent	None
30 J.F.	"		None	"	"
31 J.W.	"		Sedatives, antacids (P), Banthine (P)	"	"
33 I.K.	"		Robuden (P)	More relief	"
35 L.	"		Alkalis and sedatives (P)	Moderate relief	"
41 M.A.	"		Sedatives, Gelusil, atropine (F)	Excellent	"
43 J.B.	"		None	"	"
45 J.R.	"		Antacids (P)	"	Slight constipation
58 N.F.	"		Pro-Banthine (P)	"	Mild xerostomia
61 J. F.	"		Robuden controlled for 6 mos.	No relief in recurrence	None
68 O.K.	"		Antacids (P)	" "	"
69 M.I.	"		Banthine (P), Robuden (G)	" "	"
70 A.G.	"		None	Good	"
73 A.G.	"		Robuden (G)	No relief in "	"Sick"
75 L.R.	"		Antacids, Banthine, Robuden (all F)	" (gastrectomy)	None
76 H.L.	"		Antacids, phenob. and belladonna (F)	Excellent	"
78 L.C.			Sedatives, alkalis (P)	"	"
7 G.L.	Gastric ulcer	3 years	Antacids, phenobarb., atropine (P)	Better	"
32 J.G.	"		Robuden, well 9 mos. (E)	Recurrence controlled	"
39 L.S.	"		All forms (F)	Sl. temp. improvement	"
49 E.B.	"		Antacids, sedatives (P), Robuden (P)	Excellent—Robuden and Antrenyl	"
22 I.S.	Prepyloric ulcer		Lactol (P)	"	"
			Diarrhœas		
14 T.P.	Ulcer. colitis		Stand.—partial improvement	Additional improvement	"
15 N.R.	"		Barbit.; Mist. bism. co. (P)	Excellent	"
			Later recurrence; same treat. (P)	"	"
16 H.R.	"		None	"	"
25 I.T.	"		Standard (P)	Controlled for 1 year	"
			Later recurrence	Controlled in few days	"
27 R.L.	"	Ileostomy	Constant liquid flow; frequent bag changes. Atropine (F)	Mushy; 1-2 changes daily; used 1 year	"
18 E.S.	"	"	Same as above. Atropine (F)	Contents of bag thicker; one change daily; 1½ yrs.	"
34 N.O.	"		Robuden (P)	No further improvement	"
I R.G.	Funct. diarrhœa		Standard (P)	" "	"
6 C.K.	"		None	No improvement	"
54 L.D.	"		Bellergal (P)	No change	"
57 E.E.	"		Sed., Kaopect. (F)	Excellent	"
2 I.H.	"		Sed., belladonna (P)	"	"
42 J.A.	Acute diarrhœa		None	"	"
51 H.C.	"	"	"	"	"
56 J.E.	Post-aureomycin		Various (F)	"	Groggy and sleepy
79 M.T.	Cause?	2 wks.	None	"	None
59 J.F.	Achlorhydria		HCl dil.; Bellergal (F)	"	"
21 M.S.	Post-amœbiasis		Kaopect., barbit. and bellad., Banthine (F)	Marked improvement	Headaches with 2.5 mg. t.i.d.

G = good; P = partial; F = poor; M = mydriasis

drugs, such as cessation of peristalsis, marked delay in emptying and abolition of propulsive and segmental activity in the small bowel in most instances. They state that these effects are less striking when the drug is administered orally. The author has found orally administered Antrenyl to be extremely effective in slowing motility of the small bowel in two instances of

ileostomy instituted for ulcerative colitis and described further in the text.

The effect on perspiration was studied by Zupko and Prokop,[18] who tested the effect of four anticholinergics on 116 hidrose and 21 normal subjects. None was completely effective in controlling sweating; Antrenyl and Pamine (epoxymethamine bromide) proved to be the most effective.

In the present study an attempt was made to evaluate the therapeutic efficacy of Antrenyl (a quaternary compound, oxyphenonium bromide or the phenyl-cyclohexyl-oxyacetic acid ester of diethylaminomethyl bromide) as compared with standard forms of therapy and with other new anticholinergic drugs, in patients with peptic ulcer and other organic or functional diseases of the gastrointestinal tract. Animal studies with this drug, and studies of its effect on various digestive disorders in man, have been reported in a number of publications.[4, 15-33]

MATERIAL AND METHOD

The subjects observed in this study consisted, with few exceptions, of patients whose condition had failed to respond adequately to treatment while under our care or that of others. Of the 80 patients involved, duodenal ulcer was present in 24; gastric ulcer in five; ulcerative colitis in five; functional diarrhœa in six; acute diarrhœa in three; post-ileostomy difficulties in two ulcerative colitis patients; post-aureomycin diarrhœa in one, and miscellaneous complaints in 24.

In all cases where the previous treatment was known, the method of treatment is indicated (Tables I and II). In 65 of the patients the antecedent treatment had been entirely unsatisfactory, or the improvement obtained had not been considered adequate by the patient; in four the previous treatment was unknown and 11 had not had any. Thus each patient, with the exception of the 11 who had not had previous treatment, served as his own control.

All patients were ambulatory and remained at work. Standard diets, appropriate for each condition treated, were employed; antacids were prescribed for the ulcer patients. Antrenyl in 5 mg. doses was administered three or four times daily, except in one instance (patient No. 21) in which persistent headache necessitated reduction of the dose to ½ tablet three times daily; the headaches diminished considerably in intensity but there was no loss in efficacy. Patient No. 49 had been on antacids and sedatives for some time with only partial relief and little additional improvement with Robuden (a gastroduodenal extract); the addition of Antrenyl controlled the symptoms completely.

The temptation to increase the dose of Antrenyl, when promising but inadequate response was being obtained, was scrupulously avoided. It was felt that working with the basic pharmacological units would make comparison of results obtained with the different pharmaceutical agents employed more informative. Obviously there is no basis of comparison unless the standard basic dose of each drug is employed. There is a possibility that in those patients who obtained little or no benefit from Antrenyl larger doses might have been productive of better results.

RESULTS

The results obtained with Antrenyl are summarized in Table III. Of the 24 duodenal ulcer patients, four had not had previous treatment; of the remaining 20 only three obtained satisfactory results with the antecedent treatment employed and seven obtained partial relief. The gastric ulcer patients fared comparably, only one of the four treated patients having obtained satisfactory results with previous treatment, while of the five Antrenyl-treated gastric ulcer patients, satisfactory results were obtained in three (60%). In the entire group of ulcer patients satisfactory results were obtained in 69% of the Antrenyl-treated patients and in 16.5% of the patients treated by other methods.

Antrenyl gave satisfactory results in 18.3% of the 18 patients with diarrhœa, of whom seven had ulcerative colitis, while previous therapy had given comparable benefit in only 5.5%.

For the entire group of 80 patients, satisfactory results were obtained with Antrenyl in 67.5%; of the 65 who had had previous therapy of various types, only 10.8% had obtained satisfactory results.

The patients were not warned or questioned concerning side-reactions, on the assumption that significant reactions would be brought to the attention of the physician; these were recorded when information was volunteered by the patient. In the dosage administered, side-reactions were not at all frequent or troublesome: dribbling occurred in one patient; slight

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TABLE II.

Case No.	*Disease*	*Duration*	*Therapy prior to Antrenyl*	*Results with Antrenyl*	*Reactions*
11 C.N.	Hyperbromidrosis	3 years	Standard deodorants	Excellent	None
80 J.N.	"	10 years	" "	"	"
52 S.C.	Post-amœbiasis		Kaopect., barbit. and bellad. (F), Banthine (F)	"	"
24 M.W.	Postcholecystectomy		Varied (F)	No improvement	"
55 A.E.	Duod. ulcer, perforation; repair		Banthine (P)	Excellent	
71 L.N.	Dyspepsia; gastric polyp		Varied; no relief	No relief	
74 A.W.	Hypertrophic gastritis; prolapsed m.m.		Antacids; sedatives (F)	Partial relief	
77 A.R.	Duodenitis		Phenobarb., bellad. (F)	Excellent	"
3 C.H.	Recurrent nausea; 5 yrs.		Varied (F)	"	"
4 L.W.	Nausea; cramps		Sedat., bellad. (F)	"	"
8 H.M.	Nausea	5 years	Sedat., antacids (P)	Excellent with antacids	"
17 J.S.	Hyperacidity	2 years	Temp. relief with alkalis	" " "	"
19 B.S.	Abd. pain; intest. hypermotility		None	No relief. Relieved by bromides	"
20 K.S.	Hyperacidity; spastic colon		Fleeting aid with bicarb.	No relief. Relief with barbit. and atropine	"
29 P.T.	Upper abd. distress; neurosis		Varied; no relief (F)	No relief	"
36 M.S.	Hyperacidity		Sedatives (P)	No relief	"
37 I.M.	Irritable colon		Sedatives (P)	Excellent	"
38 A.W.	" "		Sedat. and bellad. (P)	Additional improvement	"
40 J.T.	" "		Varied (F)	No relief	"
44 S.B.	Spastic colon		Varied (F) including Sod. Amytal (F)	Progressive improvement	"
46 G.B.	" "		Bellad. and phenobarb. (F)	Only temp. relief	"
47 F.B.	" "		Sedatives (F)	Excellent	"
48 C.B.	Constip. alternating with diarrhœa		Not determinable (F)	"	"
50 J.C.	Epigastr. pain; funct.		" "	"	"
53 A.F.	Spastic colon		Sedatives; belladonna (F)	Partial only	"
60 A.F.	G.I. upsets		Banthine (F)	Poor	"
62 E.G.	Spastic colon		Unknown (F)	Excellent	"
63 I.G.	" "		Banthine (P)	"	Dryness
64 M.G.	Gen. abd. pain; funct.		Bellergal (P)	"	None
65 L.G.	L. " " "		Various (P)	Poor	"Peculiar sensation"
66 M.H.	Spast. colon; hyperacid.		Bellad. and antacids (P)	Excellent (with antacids)	None
67 E.H.	" " "		Unknown (P)	Partial	None
72 L.G.	Lower abd. pain		Sedat., bellad. (F)	Poor	Drowsy

P = partial; F = poor.

TABLE III.

Disease	*No.*	*Previous treatment* *Satisfactory**	*Partial†*	*Untreated*	*Antrenyl* *Satisfactory**	*Partial†*	*Satisfactory %*
Duodenal ulcer	24	3	17	4	17	7	71
Gastric ulcer	5	1	3	1	3	2	60
Total ulcer	29	4	20	5	20	9	69
Ulcer. colitis	7	0	6	1	5	2	
Funct. diarrhœa	5	0	3	2	4	1	
Acute diarrhœa	3	0	3	0	3	0	
Post-aureomycin	1	1	0	0	1	0	
Post-amœbiasis	1	0	1	0	1	0	
Unclassified	1	0	1	0	1	0	
Total diarrhœas	18	1 (5.5%)	14	3	15 (83.3%)	3	83.3
Spastic colon	10	2	1	7	6	4	60
Nausea	3	0	3	0	3	0	
Miscellaneous	20	0	20	0	10	10	56.5
Total cases	80	7 (10.8%)	58	15	54 (67.5%)	26	67.5

*Satisfactory to excellent. †Partial to poor.

NOTE:—Success with previous forms of therapy was achieved in 10.8% (7 out of 65 treated patients). Success with Antrenyl was achieved in 67.5% (54 out of 80 treated patients).

constipation in two; dulling of the senses in one; mild xerostomia in two; somnolence in two; headache in one, even on 2.5 mg. t.i.d.; peculiar sensations in one; and a "sick feeling" in one. The symptoms were not sufficiently troublesome in any case to warrant discontinuation of the drug.

The average age of the duodenal ulcer group was 45, with a range of 19 to 66; there were 22 men and two women. For the gastric ulcer group, of which there were five, the average age was 46, with a range of 35 to 54; there were three men and two women. The ulcerative colitis patients ranged in age from 21 to 55; there were three men and four women. In the group of 14 patients with diarrhœa not due to ulcerative colitis, there were 10 women and four men, with an age range of 28 to 60 and an average of 43.

Almost all of these patients had had trial of therapy for varying periods of time, in some instances for decades. Examination of Tables I and II will reveal the various forms of therapy employed before the use of Antrenyl. In a few instances, when the patient improved on Antrenyl, he was put back on the preceding therapy with resulting recurrence of symptoms, which were again controlled on reverting to Antrenyl. It will likewise be seen that many of the patients who improved on Antrenyl had previously tried several forms of therapy without any or without adequate relief. In a few patients Antrenyl was not effective in a recurrence, although it had given good results in the first therapeutic trial (Cases 61, 68, 69, 73 and 75).

Case 18. S., Woman aged 44. Known to have ulcerative colitis since 1935 and subjected to ileostomy in 1942. She is employed as a secretary and found the ileostomy a great hindrance since it required attention two or three times during the working day. Amphojel, Kaopectate, Cellothyl and atropine in ample doses failed completely to improve the situation. In April 1953, Antrenyl in 5 mg. doses three times daily was prescribed; it reduced the rate of flow and thickened the consistency of the stool materially. Since the institution of this treatment she has been spared the embarrassment of prolonged absence from her desk, the ileostomy now requiring attention in the morning and evening only. There has not been any tendency to loss of efficacy throughout this time, and the dose could be reduced to two tablets without loss of benefit. The patient also found that she could omit the Antrenyl for two weeks out of every two months without pronounced change in bowel function. There have not been any side-effects.

Case 27. L., woman aged 21. Ulcerative colitis since July 1950. Subjected to ileostomy in the early part of 1953. She found the care of the ileostomy very troublesome because the continuous flow of thin fæcal material made frequent bag changes difficult, inconvenient and unpleasant. The usual efforts at correcting the condition, including atropine administration, were ineffectual. Shortly after Antrenyl in doses of 2.5 mg. t.i.d. was prescribed, the fæcal consistency changed from thin fluid to mushy; the skin about the ileostomy rapidly improved. Discontinuation of the Antrenyl brought about a return to the previous status which was again quickly reversed with the readministration of Antrenyl. The patient has continued its use but finds it more efficacious in 5 mg. doses. She has not had any side-effects.

Case 52. A.O'N., man aged 25. Typical ulcer symptoms since 1951. Duodenal crater visualized radiologically in September 1952. The patient responded well to 4 tablets of Banthine daily, but complained of constipation and extreme dryness of the mouth on rising. He continued relatively well on this treatment with the periodic addition of an antacid powder containing magnesium carbonate, which served to relieve the constipation. In the spring of 1953 he complained of incomplete relief. Antrenyl, in four 5 mg. doses daily, was substituted for Banthine; the treatment otherwise remained unchanged. Within three days he was completely free from pain, and continued so during the succeeding two weeks. There was still some tendency to constipation, which was relieved by an agar-mineral-oil emulsion. On May 14, he reported that he had run out of Antrenyl and reverted to the use of Banthine, of which he had a supply, with recurrence of the pain. He was instructed to return to the use of Antrenyl, was quickly relieved of the symptoms, and has remained well since by following his dietary regimen and taking Antrenyl in the spring and fall.

Case 18. N.F., man aged 45. First seen November 6, 1953. He had been troubled with vague upper abdominal distress for some years. In October 1952, he had been placed on Pro-Banthine (propantheline bromide), to which he responded moderately well. The beneficial effect gradually wore off and this prompted him to seek further help. At this time a duodenal ulcer crater was readily demonstrated radiologically and, since Pro-Banthine was no longer effective, the patient was placed on a restricted dietary regimen, compressed milk tablets for interval nourishment, Neutrasil four times daily, and 5 mg. Antrenyl four times daily. The severity of the symptoms gradually abated. On February 28, 1954, x-ray examination revealed the presence of a deformed duodenum, but the previously visualized crater was not now present. He has remained free from symptoms to the present time.

Case 50. E.N., man aged 42. The patient was first seen in April 1947, complaining of pain in the upper abdomen during the spring and fall of the preceding year and at the time of the visit. He gave a typical history of duodenal ulcer and this was confirmed by x-ray examination. An alkaline powder with belladonna and phenobarbital in addition to a suitable diet relieved him of pain and he remained moderately well on this treatment until the spring of 1953 when business difficulties precipitated a severe recurrence. He responded well to Banthine and Nembutal (pentobarbital sodium, given by another physician) for a period of six weeks. He then discontinued the Nembutal and during the following three weeks the pain became troublesome. Antrenyl, 5 mg. four times daily, and Gelusil tablets quickly relieved his symptoms and carried him through the following fall without recurrence. He has remained well since on the above regimen.

DISCUSSION

In view of the perennially recurring reference in the medical literature to the fact that one or another anticholinergic drug does not accomplish any more than atropine when it is admin-

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istered in adequate doses, it seems desirable to attempt a clarification of this point. With certain important qualifications this statement is considered to be true, and its truth can very readily be demonstrated in acute experiments. In actual practice, however, the efficiency of atropine and belladonna is vitiated by the uncomfortably narrow margin between the level of availability to the organism of the desired pharmacological effects and the level at which undesired side-reactions occur. Presumably the effective blood concentration for both actions is very similar. The subtle, or not so subtle, difference between the various anticholinergics may lie in the degree of separation between the effective concentrations of the drug for the desired action and for the troublesome side-reactions, as well as in the receptive sensitivity of the specific organism subjected to the influence of such drugs.

Clarification of the place of atropine and belladonna in the treatment of chronic and recurring conditions would be very useful, especially in eliminating the uncertainty created in the mind of the medical practitioner as to the legitimacy of employing expensive drugs in the place of economical ones which would serve as well. While it seems to be generally accepted that atropine or belladonna alkaloids administered parenterally appear to be as efficient or nearly as efficient as the new anticholinergics, this method of administration cannot be employed except as a temporary measure, and certainly does not come into question in the vast majority of cases requiring anticholinergic therapy.

It had been the author's practice to prescribe the tincture of belladonna or a solution of atropine sulphate, starting with a basic number of drops three times daily, to increase this dose by three drops daily until xerostomia and/or mydriasis occurred, and then to continue with that dose reduced by two or three minims. This plan provided a certain measure of security against overdosage, but was not productive of confidence in effective action, since the dose of the drug was not being increased to the point of desired specific effectiveness, but was diminished before attainment of this objective because of undesirable side-effects. With newer anticholinergics, evidence of overdosage is not infrequently revealed by too great an intensity of the desired effect, such as smooth muscle relaxation as evidenced by the frequently reported urinary dribbling. A few months after the introduction of Banthine, several instances of perforation following its use in the presence of gastric retention occasioned by duodenal scarring were reported. It has also been my misfortune to have this occur in one of my cases. Presumably such perforation is precipitated by pronounced relaxation of the gastric musculature, leading to progressive increase in the intragastric pressure as a result of greater gastric retention, and the ultimate recurrent transmission of this excessive hydrostatic pressure to the ulcer-weakened area. If this degree of gastric relaxation occurs with atropine, the author has not encountered instances of it in his practice or in the literature. Perhaps one of the best indications of the difference in effectiveness of two anticholinergic drugs is to be found in the relative efficiency of Banthine and the more recent product of the same pharmacological laboratory, Pro-Banthine. If to these manifestations of difference of action is added the evidence that a patient who does not respond satisfactorily to one such drug may do better with another (several such examples can be noted in Tables I and II), the advantage of having available a number of such preparations is obvious.

It is possible that the specific biological response of tissues and organs to pharmacologically active substances is dependent not only upon the specific pharmacological action of the preparation, but also upon the receptivity of the tissues for that preparation. It is perhaps permissible to speculate that it is this variability in biological receptivity that gives rise to the divergent views concerning the efficacy of the belladonna-atropine group of preparations, and that sequential administration of different anticholinergics, after failure of one or more to relieve the patient's symptoms, may clarify the relative value of such substances, including the belladonna-atropine group. The present study has been carried out in this spirit, and it is proposed to add to this series in the hope of contributing toward the clarification of this problem.

SUMMARY AND CONCLUSIONS

1. The status of the newer anticholinergic drugs in relation to atropine and belladonna is discussed.

2. Eighty patients with organic or functional disease of the gastrointestinal tract were the subject of this study.

3. Of these, 24 had duodenal and five gastric ulcer; seven ulcerative colitis; five functional diarrhœa; three acute diarrhœa of unknown etiology; one each post-aureomycin diarrhœa, post-amœbiasis diarrhœa and unclassified diarrhœa; 10 spastic colon.

4. The treatment was ambulatory on a standard diet appropriate to the particular condition, and each patient received a 5 mg. tablet of Antrenyl (oxyphenonium bromide) four times daily.

5. When the response to Antrenyl was only partially satisfactory, in the interest of standardized treatment the dose was not increased.

6. Of the 65 previously treated patients, satisfactory results had been achieved in seven (10.8%). Assuming that all of the 15 who had not had earlier therapy would have benefited from prior medication, the percentage of satisfactory results for the whole group would rise to 27.5%.

7. Under treatment with Antrenyl, 54 of the entire group of 80 patients, i.e. 67.5%, obtained satisfactory results.

8. The substitution of Antrenyl for other anticholinergic therapy constituted a distinct therapeutic advantage for this group of patients.

9. Worthy of special note are the results in two cases of ileostomy in which the administration of Antrenyl altered the consistency of the ileal contents and increased considerably the transit time through the ileum.*

10. In this group of 80 patients, side-reactions were not troublesome and occurred in 11 patients.

*Since the above was written, the writer has had the opportunity of treating successfully a third patient with ileostomy instituted for ulcerative colitis.

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RÉSUMÉ

L'auteur revoit ici les résultats qu'il a obtenus avec l'Antrényl dans le traitement de 80 malades souffrant d'affections du tractus gastro-intestinal et compare les propriétés anticholinergiques de ce médicament avec celles de l'atropine et de la belladonne. Les troubles traités étaient en rapport avec des ulcères gastriques et duodénaux, recto-colites hémorragiques purulentes, diarrhées fonctionnelles ou d'origine indéterminée et spasmes du côlon. Les doses employées étaient de 5 mg. de bromométhylate d'oxyphénonium (ANTRENYL, marque déposée) quatre fois par jour. Les doses ne furent pas augmentées, même si les résultats obtenus n'étaient pas entièrement satisfaisants. Des 65 malades ayant été traités antérieurement, seulement 7 (10.8%) avaient eu des résultats satisfaisants. En supposant que tous les 15 qui n'avaient jamais été traités eussent subi une amélioration d'une médication antérieure quelconque, le pourcentage de résultats satisfaisants du groupe entier, sous traitement autre que celui qui nous intéresse ici, aurait été de 27.5%; or, avec l'Antrényl, 54 des 80 malades (ou 67.5%) accusèrent une amélioration sensible. On peut donc conclure que la substitution de ce médicament aux autres préparations anticholinergiques présente un avantage marqué dans le groupe que nous avons observé. Mention particulière doit être faite de trois cas d'iléostomie chez qui l'administration d'Antrényl changea la consistance du contenu de l'iléum et en ralentit beaucoup le transit. Onze malades du groupe accusèrent quelques à côté insignifiants.

M.R.D.

1956 PRIZE ESSAY CONTEST

The Council on Undergraduate Medical Education of the American College of Chest Physicians offers three cash awards to be given annually for the best contributions prepared by undergraduate medical students on any phase in the diagnosis and treatment of chest diseases (heart and/or lungs). The first prize will consist of a cash award of $250; the second, $100; and the third, $50. Each winner will also receive a certificate of merit. The winning contributions will be announced at the 22nd Annual Meeting of the American College of Chest Physicians, to be held in Chicago, Ill., June 7-10, 1956. All manuscripts become the property of the American College of Chest Physicians. Application forms may be obtained by writing to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, U.S.A.

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ECONOMIC AND SOCIAL CONSEQUENCES OF ILL HEALTH AND DISABILITY ON A NATIONAL SCALE

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THE STATE OF the nation's health in Canada continues to show steady progress and compares favourably with that of other countries. Outstanding advances in medical science, together with greatly expanded facilities for health care, have brought about this improved condition, and present an encouraging picture for the future. Enough praise cannot be given to the medical and allied health professions in Canada which through medical research and the application of improved principles of health care are playing such a dominant role in maintaining this upward trend. The National Health Grants programme is also making a major contribution in providing substantial assistance for the extension of services in the health field. All phases of health care, however, have not shown to advantage. While major advances are being made in many fields, resulting in the virtual elimination of disease conditions, improvement in other areas is less pronounced. The over-all progress, plus the selective character of the advance, is reflected by certain health statistics and will have an important bearing on future health care.

For example, the study of life expectancy tables (Table I) demonstrates that life expectancy at birth in 1951 was 66.3 years for males and 70.8 for females. This was a gain for males of 3.3 years since 1941, compared with three years in the previous decade. Females showed a corresponding gain of 4.5 and 4.2 years during the same periods.

The increases in life expectancy have been predominantly in early life, particularly in infancy, smaller gains being shown up to the middle years and very little improvement being demonstrated in advanced age. The gain in childhood and adolescence is attributed mainly to reduction in mortality from infectious diseases. This improved picture in early life through middle life is further illustrated by Table II,

TABLE I.

COMPARISON OF SURVIVALS, PROBABILITY OF DEATH AND LIFE EXPECTANCY FOR PARTICULAR AGES*

Age	*1931*	*1941*	*1951*
Males—			
0	60.00	62.96	66.33
20	49.05	49.57	50.76
40	31.98	31.87	32.45
60	16.29	16.06	16.49
80	5.61	5.54	5.84
Females—			
0	62.10	66.30	70.83
20	49.76	51.76	54.41
40	33.02	33.99	35.63
60	17.15	17.62	18.64
80	5.92	6.03	6.38

*Canadian Life Table, 1951 (Dominion Bureau of Statistics, Ottawa, 1953).

indicating the distribution of deaths by age and sex by decades from 1921 to 1951.

It will be noted that the gain is most marked in infancy and gradually tapers off and flattens out in the forties. After the forties, while female deaths continue to show a downward trend, male deaths either increase or at best remain at about the same level. This unfavourable picture as far as males are concerned may be due to a variety of factors, which might well include certain environmental stresses which are more frequent and severe for males. These statistics, with information from other sources, support the contention that while many acute diseases have bowed to medical progress, the chronic, crippling and degenerative conditions have not been as amenable to treatment. In addition, many of these disabilities are commonly associated with later life, and as life expectancy is on the increase we will be more and more concerned with their control. Medical progress, by skimming off the acute and more sensational diseases, has laid bare the most challenging problem for the future—the control of chronic and crippling disease. In terms of social consequences, this area is bound to dominate the health field.

Progress in medicine has resulted in a great increase in medical knowledge, calling for an increase in specialist services. Some would say that there has been over-specialization, but, in any case, there is now concerted action within the profession to ensure better coordination of effort between general practitioners and specialists. While the medical profession has always accepted a broad responsibility for the health

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TABLE II.

DISTRIBUTION OF DEATHS BY AGE AND SEX (RATE PER 1,000 POPULATION)*

Age group	1921		1931		1941		1951	
	Male	Female	Male	Female	Male	Female	Male	Female
Under 5 years	28.9	22.7	26.8	21.2	20.0	15.5	11.1	8.8
5 - 9 years	3.1	2.7	2.2	1.7	1.7	1.3	1.0	0.7
10 - 14 "	2.1	1.9	1.5	1.5	1.4	1.0	0.8	0.5
15 - 19 "	3.1	2.7	2.5	2.2	2.0	1.5	1.3	0.9
20 - 24 "	3.7	3.7	3.2	3.2	2.6	2.0	1.9	1.0
25 - 29 "	4.0	4.1	3.4	3.8	2.7	2.5	1.8	1.1
30 - 34 "	3.8	4.5	3.5	4.2	2.8	2.8	2.1	1.5
35 - 39 "	4.7	5.5	4.2	4.8	3.8	3.4	2.5	2.0
40 - 44 "	5.6	5.9	5.4	5.0	5.0	4.5	3.9	3.0
45 - 49 "	7.3	7.1	7.2	6.6	7.3	6.0	6.3	4.5
50 - 54 "	9.8	10.2	10.7	9.0	10.6	8.1	10.3	6.4
55 - 59 "	15.2	13.5	15.4	13.4	16.0	12.3	16.2	10.2
60 - 64 "	21.9	19.7	22.9	20.7	24.2	18.5	24.4	16.1
65 - 69 "	33.4	33.2	35.2	30.3	37.3	30.4	35.1	24.9
70 - 74 "	56.9	52.8	55.0	49.1	58.5	47.0	54.5	41.6
75 - 79 "	89.4	80.9	87.4	82.9	95.7	79.7	87.7	73.3
80 - 84 "	133.8	122.4	134.1	127.1	147.6	131.2	135.6	120.6
85 years or over	228.2	224.9	228.1	212.6	241.9	229.3	234.8	211.9

*Canada Year Book, 1954, Dominion Bureau of Statistics, Ottawa.

care of individuals, this increase in specialization, combined with a shortage of medical personnel and other factors, makes it difficult for the modern physician, particularly in our larger communities, personally to ensure social re-establishment of patients. If it is accepted that the profession cannot provide for this broad concept of care, it would appear that the time has come when the medical profession should formulate principles for establishing a team approach which would make full use of the skills of other professional groups. These principles should be developed on the basic concept that team leadership lies with the medical profession.

The question of coordinating and integrating the services of other professional groups also highlights the need for a more rational utilization of the numerous types of present-day specialized facilities which are also an outgrowth of specialization. The complexities of modern health care have resulted in the establishment of varieties of diagnostic centres, in the setting up of hospitals for chronic cases, in an increase in hospitals for acute cases, and in specialized institutions and clinics, not to mention the growth of facilities for domiciliary care. Frequently, these units are set up without regard to any over-all plan. This feature was appreciated at the First National Conference on Rehabilitation of the Disabled, when the Medical Sub-Committee strongly recommended that rehabilitation services should be carefully coordinated with the hospitals and other health arrangements for the particular area or community. Since this report was first submitted, it has been used extensively in discussions with the provinces in connection with the development of their rehabilitation plans. I commend this document as one which appears to set out general principles which can be applied across our country.

In dealing with this broad subject of economic and social consequences of ill health and disability on a national scale, it is only possible in a paper such as this to touch on a few of the highlights. I propose therefore, during the remainder of this article, to deal with four aspects which seem to me to be important, in the hope that these items will serve as a stimulus for subsequent discussion covering broader areas. The subjects which I propose to discuss are as follows: (1) our national health bill, (2) the effect of disability on productivity, (3) permanent physical disability in Canada, and (4) our changing health picture and the related social problems.

1. OUR NATIONAL HEALTH BILL

It is estimated, from information taken from the Canadian Sickness Survey and other sources, that the total health bill for Canada in 1951 was $675,000,000. This includes expenditures for personal health care and treatment and for public and other technical health services, and capital expenditures, and includes payment by governments. On a population basis, it represents a

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yearly expenditure of about $45 per person. It was further estimated that in 1952 this total expenditure had risen to $760,000,000. Of the total expended in 1951, it was estimated on the basis of the Canadian Sickness Survey that a sum of $373,800,000 was actually spent by individual family units for the purchase of health services. It will be noted from Table III that this amount includes expenditures for prepayment plans and direct expenditure. Prepayment plans account for 23.7% of the total, while direct expenditure covers the balance. Included in the direct expenditure is payment for services, namely to physicians (23.5%), hospital care (12.4%), dental services (8.8%), eye services (3.8%), nursing care (3.1%) and combined bills (2.8%). Drugs and appliances account for 20.1% of the total.

TABLE III.

ESTIMATED TOTAL EXPENDITURES ON HEALTH IN 1951*

	Estimated expenditures $	*Percentage of total*	
Total expenditures	373,800,000	100.0	
Expenditures for prepayment plans:			
Medical care	11,800,000	3.2	23.7
Hospital care	43,500,000	11.6	
Combined plans	33,100,000	8.9	
Expenditures made directly for:			
Services			
Physicians' services	87,700,000	23.5	54.4
Hospital care	46,500,000	12.4	
Dental services	32,900,000	8.8	
Eye services	14,100,000	3.8	
Nursing care—home	3,900,000	1.0	
—hospital	7,800,000	2.1	
Combined bills	10,300,000	2.8	
Drugs and appliances—			
Medicine—prescribed	46,100,000	12.3	20.1
—not prescribed	26,900,000	7.2	
Appliances and equipment	2,300,000	0.6	
Other	7,000,000	1.9	

*Canadian Sickness Survey 1950-51, Dominion Bureau of Statistics, Ottawa, May 1953.

In addition, it must be remembered that the above costs do not include the income maintenance programme related to disabled persons. These expenditures may be roughly estimated to be something over $200,000,000 annually. They include programmes such as veterans' pensions, blindness pensions, workmen's compensation, disability allowances, and a substantial part of the veterans' allowance programme, the mothers' allowance programme and old age assistance. A new development in the field of income maintenance assistance for disabled persons is the proposed permanent and total disability programme, which is being jointly sponsored by the federal government and the provinces. This will add to the bill now being paid for disability assistance. In connection with the last-named programme, it should be mentioned that emphasis is being placed on a close tie-in between the pensions programme and rehabilitation.

To summarize, it will be seen that our total national health bill is somewhere in the neighbourhood of a billion dollars a year, and this for a population of some 15,000,000. These figures represent a substantial outlay for the health care of our people and this is bound to have a considerable economic as well as social impact. Any procedures which will decrease the duration and severity of illness in a substantial proportion of cases will effect a major national saving.

2. EFFECT OF DISABILITY ON PRODUCTIVITY

Disability can be broadly divided into two categories: temporary and permanent. An indication of the effect of temporary disability within the civilian labour force is given by information from various labour and industrial sources which show that there is an average absenteeism rate for illness per employed person of about 9½ days per year. Similarly, in a total labour force of over five million, information from the Dominion Bureau of Statistics indicates that an average of about 50,000 persons are away from work each day because of illness, or that 0.9% of the total labour force is incapacitated on this daily basis. It will be seen from these figures that even a reduction in absenteeism for illness of one or two days a year for each worker would add substantial amounts to our national economy. It is of interest to note that the Canadian Sickness Survey indicates that over half of all the diseases reported during the Survey were diseases of the respiratory system; in fact, the common cold and influenza represented 45% of the total of all conditions.

When permanent disability is considered, the results of the Canadian Sickness Survey provide a valuable index of the effect of permanent physical disability on productivity. In this study, the preliminary findings show that almost one-half of the 423,000 severely or totally physically disabled persons in Canada were supported by their immediate families or other relatives. More than one in six depended solely on a public pension (old age pension, blind pension, pension for war disability, workmen's compensation, mothers' allowance). Only about one out of seven of the severely disabled reported income

from employment. This picture could be radically changed by the application of good principles of rehabilitation.

3. PERMANENT PHYSICAL DISABILITY

It may be of interest to summarize the findings developed from the Canadian Sickness Survey in connection with permanent physical disability in Canada.

The estimated number of persons in Canada suffering from a permanent physical disability of some degree at the time of the survey was about one million. Of these, an estimated 423,000 persons were severely or totally disabled, including 236,000 aged 18 to 64, or about 3% of the population in this age group.

About 99,000 persons were estimated to be totally disabled. Diseases of the heart and arteries, impairment due to accidents, arthritis or rheumatism, diseases of the nervous system, deafness and blindness, in that order, accounted for about 58% of all primary permanent physical disabilities. Other causes of primary disability, each affecting approximately 3% of the disabled population, were tuberculosis, chronic eye diseases, congenital malformations, asthma, and varicose veins. Impairment due to poliomyelitis, peptic ulcer and neoplasms, each accounted for about 2% of the disabled population.

Of all disabled persons, about two out of five reported more than one type of permanent disability.

Some 57,000, or 14%, of severely or totally disabled persons had suffered from their primary disability for less than one year; 110,000 (26%) for from one to four years; 92,000 (22%) for from five to nine years, and 160,000 (38%) for ten or more years.

These figures present a challenge to the medical profession and other disciplines concerned with the development of rehabilitation in Canada.

4. OUR CHANGING HEALTH PICTURE AND THE RELATED SOCIAL PROBLEMS

From some of the statistical data presented in this paper, it will be noted that as a result of the increase in life expectancy the size of the old age group in the population is steadily increasing. This increase is due in no small part to the effective control and virtual elimination of many acute catastrophic diseases. The health picture which emerges as a result of these changes is one of greater emphasis on long-term chronic and crippling conditions. These illnesses are frequently progressive and are commonly associated with prolonged periods of illness with varying degrees of residual disability.

Loss of functional capacity can obviously have many social effects on the individual. Because he cannot pursue his former social and vocational activities, he may need considerable assistance in restoring some semblance of a balanced life. His earning power may cease or be reduced, which will add to the problem of adapting his way of life to the new conditions imposed by his disability. He may become much more dependent in every way on his family or upon community institutions and services. In the case of many of our older citizens and others in whom family ties are shaky or non-existent, the problem of providing for an adequate social life falls heavily on the community.

In addition to the medical problem, the changing social pattern of this modern age imposes other problems. The large family home in which the disabled and elderly could once be maintained has all but disappeared. Families, particularly in urban areas, are living in smaller dwellings or apartments and are no longer able to give shelter to the ill or the old. The communities in which they live are growing larger and becoming more and more impersonal.

The total effect of this changing health picture and its related social problems is a cumulative one on individuals, communities, provinces, and the nation as a whole. While acute conditions present their social problems, chronic illness, particularly in the older age groups, is likely to present the greatest social health problem in the future.

CONCLUSION

An attempt has been made to bring out a few of the important economic and social consequences of ill health and disability. Rehabilitation has much to offer in minimizing the effects of both acute and chronic illness. Medical leadership is essential to a good, well-balanced, rehabilitation programme.

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THE "M.C. NUMBER" OF SERUM IN INFECTIOUS HEPATITIS*

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IN INFECTIOUS HEPATITIS, electrophoretic identification of the various serum protein fractions shows that there is a characteristic alteration in their respective concentrations. Liver production of serum albumin is reduced, and thus an early fall in total serum protein may appear. Circulating globulins then increase, first alpha globulins (as with any fever), then beta globulins, and most notably gamma globulin. Thus during the course of the infection the albumin/globulin ratio may be reversed, from a normal mean of 1.78 to 0.75 or lower. During convalescence, globulins drop slowly with an increase in serum albumin until normal proportions are reestablished. Fig. 1 indicates these changes in a typical case.

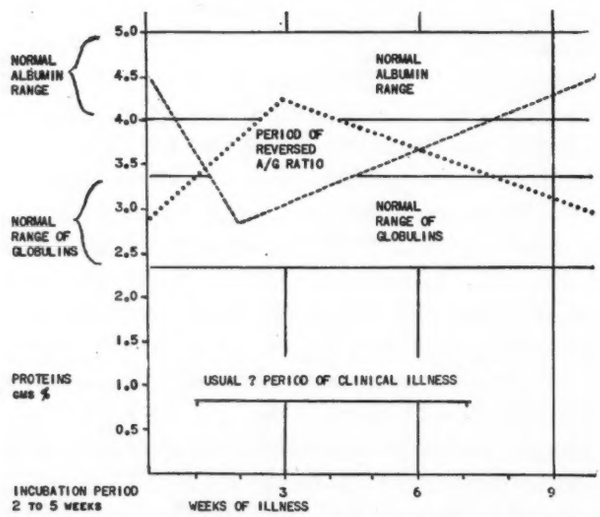

Fig. 1.—Serum albumin/globulins in infectious hepatitis estimated by electrophoresis.

	Normals	*At 3 weeks*	*At 6 weeks*	*At 9 weeks*
Bilirubin	Nil	2.0 +	0.5 +	Nil
Alk. phosphatase	3-13 units	25.0 units	20.0 units	10.0 units
A/G ratio	1.78	0.7	1.0	1.6
Ceph. cholesterol	1+	4+	2+	1+
Thymol turbidity	2½ units	6+ units	3+ units	2+ units
M.C. number	Zero	70	50	30

Broken Line—Changing concentration of serum albumin.
Dotted Line—Changing concentration of total globulins during infectious hepatitis.

Biochemical determinations of serum bilirubin, alkaline phosphatase, total proteins, and A/G ratio, are of value as laboratory aids, but electrophoretic study of the precise concentrations of the various serum proteins is not practical as yet in most cases of infectious hepatitis. Thus, empirical, non-specific flocculation tests of serum, which appear to depend for their reactivity chiefly upon increased gamma globulin, have use as rough measures of disturbance in protein metabolism. They can sometimes help to distinguish jaundice due to mechanical obstruction of bile ducts and without change in protein proportions of serum (which is non-reactive on test) from jaundice due to parenchymal damage to the liver, in which changes in serum proteins produce flocculation of test reagents.

The two flocculation tests which have found widest favour make use of reagents of different types; the cephalin-cholesterol suspension (of Hanger) is colloidal in nature, the thymol-turbidity test (of Maclagan) employs a saturated solution of that organic material. Of a third type, many tests have been designed depending upon inorganic divalent metal salts in concentrations much below that required to cause complete precipitation of proteins from solution. The first of these, in 1925, was that of Takata and Ara[2] in which mercuric chloride was used. All these are test-tube methods requiring considerable time for reaction.

During the typhus epidemic in Persia in 1943 one of us (L.E.), observing the flocculation of platinic chloride in early typhus serum, described this in Sachs's[3] account of that outbreak. Following that, observations were continued of the reactions of different metal salts with sera in various disease conditions, until the present reagent mixture, to be described, of mercuric chloride with sodium citrate and congo red was devised. This gives an immediate macroflocculation reaction with abnormal serum in suitable drop proportions on a colour reaction plate. Like the other flocculation tests of its kind, its reactivity depends principally upon increase in serum gamma globulin; albumin tends to inhibit flocculation of the reagent. The addition of congo red to the mixture is logical in consideration of its parallel ability, as described by Steabben,[4] quoting Joel, of being sensitized to flocculation by globulins while being protected from such reaction by albumin.

The relative insensitivity of the Takata-Ara reaction in a test-tube method was pointed out by Magath,[5] and Maclagan[6] stressed the real need

*From the Division of Laboratories of the Ontario Department of Health, presented to the Laboratory Section of the Canadian Public Health Association, Montreal, December, 1954.

for some stabilization of serum flocculation tests. The "M.C. number" (mercuric-chloride number) of serum, as determined by flocculation of the "M.C. reagent" (mercuric chloride reagent) described herein, measures a percentage range of reactivity with abnormal serum, and has proved most sensitive in liver dysfunction, comparable to presently accepted methods, and somewhat more useful than these in prognosis. Infectious hepatitis is an epidemic disease of armies, and most observations with M.C. reagent were made in the East during the war of 1939-45, but the disease is of constant concern to civilian populations both young and adult. Clinical laboratories can be of most service to the physician, in the investigation of liver disease, by offering a broad battery of urine and blood analyses and precise estimations of known liver functions, but it is felt that the flocculation tests have established themselves as useful, if empirical, simple laboratory procedures, and that the M.C. number of the serum represents a valuable additional item to present standard methods.

MATERIALS

Preparation of M.C. reagent requires chemicals of good reagent grade and water preferably double glass distilled. The formula is: mercuric chloride 0.2%, trisodium citrate 0.4% and congo red 0.02%. Each salt is first dissolved separately in triple strength in a suitable volume of water, conveniently 100 ml. each, that is, in concentrations 0.6%, 1.2% and 0.06% respectively, and then the second and third solutions are added to the first in equal volumes, to make up, say, 300 ml. of reagent mixture. M.C. reagent is then incubated for 24 hours at 37° C. to ripen, when the colour of the indicator becomes a clear limpid blue and the pH of the reagent mixture stabilizes at approximately the isoelectric point (pH 6.6) of gamma globulin. If stored in a refrigerator below 10° C. but above freezing, buffering effect is well maintained for months, and the mixture remains a clear blue without sediment. It should be kept preferably in glass, without contact with metal, and protected from light. Nothing should be dipped into a stock supply, but volumes poured from it as required. One rough check of a satisfactory batch of M.C. reagent is that an equal drop volume of N/10 NaOH should not produce apparent change in colour or clarity, whereas one drop volume of N/10 HCl, added initially, will produce an immediate blue precipitate, which redissolves upon the addition of a neutralizing equal drop of the alkali, with colour change of the indicator to a clear pink. For a screen test of a group of sera for routine examination, M.C. reagent is used full strength, and a normal serum, which does not show flocculation in test proportions with full strength reagent, is reported as M.C. number zero. Naming the "M.C. number" of serum was in reference to the interaction of relatively colloidal hydrophobe/hydrophil solutions as described by Freundlich.[7] For determination of the M.C. number of an abnormal serum, which does show flocculation in the screen test, percentage dilutions of M.C. reagent, conveniently nine, are made with an aqueous solution of congo red 0.02% in freshly distilled water (see Table I). These also are incubated for 24 hours at 37° C., in order to ripen solutions, fix the colour of the indicator in a limpid blue of equal intensity for all, and stabilize pH of the dilutions. Kept stoppered conveniently in glass tubes in a rack and refrigerated, they are satisfactory for use for at least one week, but dipping into a solution may tend rather rapidly to introduce change in its sensitivity in case any extraneous material is introduced.

TABLE I.

DETERMINATION OF THE "M.C. NUMBER" OF SERUM IN DROP VOLUMES ON A COLOUR REACTION PLATE

Dilutions of M.C. reagent (percentage dilutions)		*Proportions employed in test*	*Corresponding M.C. number of serum*
	Reagent diluent	*Reagent : Serum*	
Full strength		Vols. 3 : 1 Vol.	"Screen test" Zero
Parts	Parts		M.C. number
90	10	Vols. 3 : 1 Vol.	10
80	20	3 1	20
70	30	3 1	30
60	40	3 1	40
50	50	3 1	50
40	60	3 1	60
30	70	3 1	70
20	80	3 1	80
10	90	3 1	90

If the "screen test" with full-strength M.C. reagent does not show flocculation, a test serum is reported as M.C. number zero. The M.C. number of an abnormal serum is that highest percentage dilution of M.C. reagent which shows flocculation in test proportions with the test serum.

Glassware and apparatus requirements, which are minimal, include: 1. *Pipettes.* As a pipette-assembly, the barrel of a tuberculin syringe, with teat, and a long hypodermic needle (of suitable number) are most useful. The size of a drop of fluid delivered is almost entirely dependent upon the outside diameter of the nozzle, as shown by Fildes,[8] and the shaft of a hypodermic needle can be scored and broken flush, with an acceptable uniform circumference to deliver consistently a reasonably standard size drop. Of water at room temperature, needles Nos. 16, 18, and 20 deliver respectively approximately 0.03 ml., 0.02 ml. and 0.014 ml., when cut thus horizontally, and *when held vertically* in delivery. It is practical also to use a glass Pasteur capillary pipette, with teat, cut to suitable nozzle size, by reference to an American standard wire gauge, of which, for example, No. 14 (0.16 cm.) provides a nozzle of such diameter that, if the capillary tube is drawn to be as nearly circular as possible, it will deliver, *when held vertically,* a drop approximately 0.03 ml. All such pipettes of any desired nozzle size should be checked for delivery per ml. before use; if different ones are being used to handle reagent and serum separately, they should deliver drops of comparable size of the same fluid. (N.B.: The differential in size of drop between aqueous solutions and serum of normal viscosity is so slight as to be disregarded.) Such pipettes are very easy to use; glass ones, being expendable, are also perhaps pleasantest to employ since filling and complete discharge are visible.

2. *Colour reaction plates* are used for many spot tests in chemistry, and may be of the depression type with 12 depressions in a white porcelain tile (e.g. Coors). Preferable for determination of the M.C. number of serum is a heavy flat white glass (vitrolyte) or porcelain tile (useful also with any coloured antigens or in blood-bank work), with 20 red ceramic rings each of diameter 14 mm.,* arranged in pattern, two pairs of rows of five

*Available from Cutler Brands, 118 Geary Avenue, Toronto, Ontario, and from Progressive Laboratory Supplies, Jamaica, N.Y., U.S.A.

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rings each, that is, two sets of ten rings, to permit the full range of nine M.C. reagent dilutions (as well as the initial screen proportion with full strength reagent) to be observed on two specimens of serum on the same colour reaction plate. Some workers may prefer to observe flocculation reactions on clear glass slides, and of these the most satisfactory are also those with red ceramic rings, arranged in the same pattern as for the white glass plate.* A plain glass plate is also available with the ring-surface white, as background for the test, while providing some translucency.

3. *Rotator*: Agitation of the reagent-serum mixture is necessary (as it is for antigen/antibody reactions), and this can be done by rotation by hand of the colour reaction plate on the laboratory work-bench. A Boerner type rotator serves well at slow speed, but perhaps most satisfactory is a "multiple-angle-rotator"† specially designed for serological slide tests (or blood-bank work), which operates at 33 r.p.m., and the motion of which simulates hand motion of rotation and tilting, as when a slide or plate is held up for visual observation of drop reactions.

METHOD

Procedure: Glassware must be chemically clean and serum be clear, without contamination or hæmolysis. Of serum for test, one drop volume, of suitable size, is delivered from a Pasteur pipette or needle-assembly, held vertically, into each of ten depressions or rings of the colour reaction plate chosen. With the same pipette, after rinsing with distilled water (or with a separate pipette of identical nozzle size), *three* equal drop volumes of M.C. reagent full strength are delivered into the first serum drop on the plate, and *three* equal drop volumes of each of the nine M.C. reagent dilutions of Table I, in order, are delivered into the other serum drops respectively. A test total of four large drops (e.g. 0.03 x 4 = 0.12 ml.) is suitable for a depression plate in each proportion, but a ceramic ring on a white glass plate cannot retain, when rotated, more than four smaller drop volumes (at the most, 0.02 x 4 = 0.08 ml.). The ratio of *three* volumes of reagent (full strength or in any dilution) to *one* volume of test serum is standard throughout a determination of the M.C. number of an abnormal serum. These are mixed and spread, if necessary but rarely, with a glass stirring rod, and the colour reaction plate is rotated by hand or by machine for *four* minutes. Flocculation of blue granules, if present in the drop mixtures, is visible by naked eye or use of a hand lens. *The M.C. number of an abnormal serum is that highest percentage dilution of M.C. reagent in which flocculation occurs in test proportion with the test serum.* A normal serum should not show flocculation with full-strength M.C. reagent in test proportion, and its M.C. number is recorded as zero. The M.C. (mercuric chloride) screen test of many sera in a group is practical (each in single proportion only), and any serum which shows flocculation in the screen test is retested to determine its actual M.C. number. It is obvious that percentage dilutions of M.C. reagent can be prepared at any level from zero to 99%, and the nine dilutions recommended have been chosen as convenient levels only, between which a more precise M.C. number of a test serum can be determined by intermediate dilution if desired (for example, at 67% dilution, when a test serum has been found to be reactive on initial test at M.C. number 60, but not at 70).

*Available from Cutler Brands, 118 Geary Avenue, Toronto, Ontario, and from Progressive Laboratory Supplies, Jamaica, N.Y., U.S.A.

†Available from Canadian Laboratory Supplies, 3701 Dundas St. West, Toronto.

(Both white glass colour reaction plate and multiple-angle-rotator designed by the Division of Laboratories, Ontario Department of Health.)

DISCUSSION

The Central Laboratory of the Division of Laboratories of the Ontario Department of Health supplies, on special request and on an experimental basis, photographs and report of the electrophoretic analysis of serum proteins of interesting clinical specimens, particularly ones related to problem diagnoses. Electrophoretic findings are represented in Fig. 1 of the uncomplicated course of an individual case of infectious hepatitis, as considered arbitrarily in three periods of three weeks each. During the early acute period, serum albumin concentration drops, with a compensatory rise in total globulins, possibly producing a reversed A/G ratio. With or without the appearance of jaundice, flocculation tests show early reactivity, apparently reflecting the abnormal serum protein pattern which persists during the height of the infection. With normal body response, abnormal proteins then

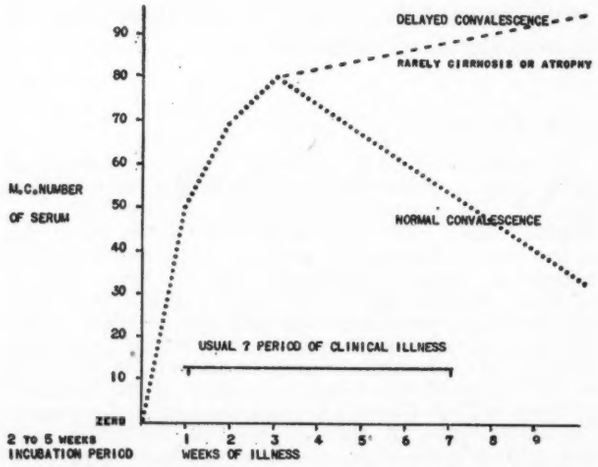

Fig. 2.—Course of the M.C. number of serum in infectious hepatitis.

NOTE:—Dotted Line—Changing M.C. number of serum during the course of an individual case of infectious hepatitis, showing characteristic range of reactivity.
(I) Early marked rise above normal, zero, as early as other flocculation tests.
(II) Slow fall during recovery, prolonged beyond the reactivity of other tests;
(III) A persistent high level in failure of the liver to return to normalcy.

begin their return to normal concentrations, until by the end of the sixth week the A/G ratio has usually approached unity. Convalescence is often prolonged beyond the ninth week, but during the third period, with adequate control of the infection, the A/G ratio returns to a relatively normal figure. In delayed recovery, persistent reaction of flocculation tests reflects a patient's continuing ill health, and the M.C. number of the serum remains at a high level. Fig. 2, a graph of the course of the M.C. number of the serum in a case of infectious hepatitis, should be considered in comparison with Fig. 1.

TABLE II.

COMPARISON OF THE M.C. NUMBER OF SERUM WITH TWO STANDARD FLOCCULATION TESTS

C.C.	*T.T.*	*M.C.*	*Specs.*	*"False" negative*	*"False" positive*	*Totals*
+/++	+/++	+/++	40			18.4% agreements positive
+/++	+/++	—	16	7.3%		
+/++	—	+/++	17	7.8%		
+/++	—	—	20		9.2%	53.2% disagreements
—	+/++	+/++	17	7.8%		
—	+/++	—	25		11.5%	
—	—	+/++	21		9.6%	
—	—	—	62			28.4% agreements negative
Totals			218 specimens			100%

Readings of tests:	Negative (—)	Positive (+)	Positive (++)
C.C. cephalin-cholesterol	Below 1+	1+	Above 1+
T.T. thymol turbidity	Below 2½ units	2½ to 5 units	5 units and above
M.C. M.C. number of serum	Zero	10 to 50	Above 50

NOTE:—These specimens were examined in the Chemistry Section of the Central Laboratory during the years 1951-52-53, as received routinely from the medical profession by mail, without access in many cases to clinical data, but for investigation of liver dysfunction.

Comparative Performance of the Three Tests Showing Graded Reactivity:

C.C. ++ 57, + 36 } 93 (42.7%), — 125

T.T. ++ 32, + 66 } 98 (44.9%), — 120

M.C. ++ 52, + 43 } 95 (43.6%), — 123

A comparison of the M.C. number of the serum with the two most commonly employed flocculation tests, the cephalin-cholesterol and the thymol-turbidity, is shown in Table II, which records 218 specimens of serum from cases of suspected liver dysfunction. These were routine examinations in the Chemistry Section of the Central Laboratory during the years 1951-52-53. Textbooks state that flocculation tests can be expected to be found reactive in more than 90% of cases of actual parenchymal, hepatocellular damage. The lowest levels of positive reactivity of the two standard tests were named in consideration of the test methods and controls employed. The large percentage of disagreements among results of the three flocculation tests can be taken as a reflection of the different natures of the test reagents—colloidal, organic and inorganic—but in general the mechanism of reactivity of all, as proven by electrophoresis, appears to be associated with abnormal serum protein patterns.

Table III compares the M.C. number of the serum with the erythrocyte sedimentation rate. Among 187 cases of active pulmonary tuberculosis, every serum specimen showed a raised E.S.R. and an increased M.C. number. Among 112 cases of infectious hepatitis (with jaundice), the E.S.R. remained within normal limits in one-third, and a further one-half showed only moderately raised E.S.R., but all sera showed an increased M.C. number.

The flocculation tests are, of course, nonspecific and may show reactivity in many conditions. In some acute infections, particularly a rapidly debilitating one like the rickettsial infection, classical typhus, marked serum protein changes occur early, and an increased M.C. number of serum was found to precede other clinical and laboratory signs. In chronic infections, particularly ones in which marked hyperglobulinæmia is a constant finding, like the tropical parasitic systemic infection kala-azar, the M.C. number of serum was consistently high. The findings for the M.C. number of serum in active tuberculosis are illustrated in Table III, and such findings can be expected in any general systemic condition, such as malignancy with cachexia, which may produce an abnormal serum protein pattern. Syphilis in advanced stages can produce such findings, and M.C. reagent is sensitive also to protein changes in spinal fluid. (Determination of the "M.C. Number of Spinal Fluid in Syphilis" will be described elsewhere.) On the other hand, cases occur in which the flocculation tests are of no clinical aid, serum remaining non-reactive in the presence of normal albumin/globulin ratio, in which albumin is in sufficient concentration to inhibit flocculation of reagent by abnormal globulins. Among apparently healthy military

men in India, 3.2% of British service personnel and 9.1% of Indian troops showed a moderately increased M.C. number of serum, that is, over 90% of all supposedly normal persons had a M.C. number of zero.

In infectious hepatitis it is felt that the sensitivity of the M.C. number of the serum is of most value in prognosis, as illustrated in Fig. 2, which should be considered in comparison with Fig. 1. In India and the Middle East during the war of 1939-45 it was often found in the course of epidemic infectious hepatitis, with or without jaundice, that, following hospitalization, with return of all other clinical and laboratory signs to normal, a soldier was discharged to duty only to find that he could not carry on and must be readmitted to hospital. The M.C. number of serum, which had been increased during the initial course of the infection, would be found to be persistently high and clinically the patient would show the picture of relapse. We are finding today that a persistently high M.C. number of serum after infectious hepatitis is confirmed by a continuing abnormal serum protein pattern on electrophoretic analysis. This may be associated with a carrier state which will not be proven until virologists are able to isolate viruses A and B of viral hepatitis. It would seem logical, however, as recommended by Stokes *et al.*,[9] that clinicians, and particularly those in charge of blood banks, might consider the advisability of placing some credence in serum reactivity to "liver function flocculation tests" of convalescent and donor bloods.

Chief laboratory advantages of the test for the "M.C. number of serum" include: (1) use of a simply prepared, relatively stable, inorganic reagent mixture self-buffered at approximately the isoelectric point of gamma globulin; (2) ease of immediate macroscopic slide test determination; (3) consistent reproducibility; (4) percentage range of reactivity.

TABLE III.

COMPARISONS OF THE M.C. NUMBER OF SERUM WITH ERYTHROCYTE SEDIMENTATION RATE

E.S.R. mm./one hour (Wintrobe)	*Infectious hepatitis (with jaundice) 112 cases—percentage distribution M.C. number of serum*					*Active tuberculosis (pulmonary) 187 cases—percentage distribution M.C. number of serum*				
	Zero	*Below 30*	*40 to 60*	*70 plus*	*Totals*	*Zero*	*Below 30*	*40 to 60*	*70 plus*	*Totals*
Below 5	0	18	14	0	32%	0	0	0	0	0%
5 to 19	0	16	32	6	54%	0	14	16	2	32%
20 to 35	0	3	5	4	12%	0	14	33	12	59%
35 plus	0	0	0	2	2%	0	2	6	1	9%
Totals	0%	37%	51%	12%	100%	0%	30%	55%	15%	100%
NOTES:	Every case of infectious hepatitis (with jaundice) showed increased M.C. number of serum, *but* one-third of all cases showed normal E.S.R. and a further one-half showed E.S.R. moderately increased only.					In active tuberculosis (pulmonary), as representing a general systemic disease with cachexia, cases showed approximately equal reactivity in both increased M.C. number of serum and increased E.S.R.				

N.B.:—Test results recorded above relate to specimens from individual patients collected and examined at the same time, but without reference to the respective stage of infection.

SUMMARY

Determination of the "M.C. number of serum" is described. The percentage range of the M.C. number of serum is discussed, with reference in particular to infectious hepatitis, and the importance of the M.C. number of serum in assisting the clinical estimation and prognosis of liver damage is stressed.

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STAPHYLOCOCCAL PNEUMONIA IN INFANCY

A REVIEW OF 40 AUTOPSIED CASES

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ANTIBIOTICS and sulfonamides have greatly reduced the infant mortality due to acute pulmonary diseases, but recently very severe and often rapidly fatal staphylococcic infections of the lungs and pleura have occurred with an apparently increasing frequency in infants. This seems to be the consequence of a wide dissemination of highly virulent and antibiotic-resistant strains of staphylococcus.

Clinical and pathological aspects of staphylococcal pneumonia in infancy have been accurately described by many authors.[1-4, 6-9] All insist on the hæmorrhagic, necrotic and suppurative nature of the pulmonary lesions, on the frequency of pleural involvement, and on the rapid and often fatal course of the disease, especially in younger infants.

The material for this study covers 40 cases with autopsy observed from 1951 to 1954 inclusive in three general hospitals in Quebec City (Enfant-Jésus hospital, 28 cases; Hôtel-Dieu hospital, 7 cases; St. François d'Assise hospital, 5 cases).†

The organism was identified bacteriologically in about half of the cases. The others were included in this series because of the gross and histological features of the lesions and the presence of numerous clumps of Gram-positive cocci in the microscopical sections. The pathological characters of the bacteriologically proved cases were so clear that the use of such criteria virtually assured the inclusion of only true staphylococcic infections and of practically all the cases seen during the period studied.

Sex.—There was no significant variance as regards sex; 22 infants were male, 18 female.

Age.—The age of the 40 infants varied from four days to twelve months, but the majority were in their second, third or fourth month of life.

Incidence.—The material was collected in three hospitals in which the relative number of children hospitalized and the autopsy rate (grossly 50%) have not significantly varied during the four years reviewed. Nevertheless the number of autopsies of cases of staphylococcal pneumonia showed a steady increase from 5 cases in 1951 to 8 in 1952, 11 in 1953 and 16 in 1954. These figures do not take into account the cured or non-autopsied cases. If they may not reflect the true incidence of the disease, they at least indicate a definite increase in its severity.

If the 40 cases reported are classified according to the time of year when autopsy was performed, there appears a definite predominance during the winter months, a lower incidence in the spring and a practical absence during the summer months (Fig. 1). The fact that no deaths were

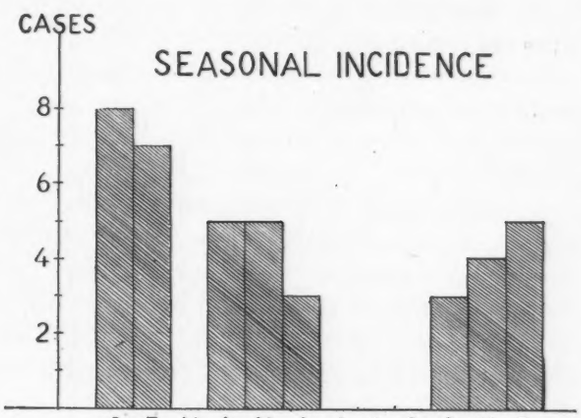

Fig. 1

reported in March is most probably explained by the small number of cases studied, but it is no doubt significant that in the three hospitals over a period of four years not a single case of staphylococcal pneumonia came to autopsy in July, August and September. The particular occurrence of the disease in winter corresponds with the general pattern of respiratory infections in the cold climate of this region.

Staphylococcal pneumonia has been reported as a complication of influenza, especially in adults.[5-10] In infants this association is certainly much less common. In only a few of the 40 cases under study do the clinical records mention an influenza-like infection before the pulmonary syndrome. But, the seasonal incidence being the same in both diseases, it may be suspected that a number of influenzal infections have passed unnoticed, especially when the first symptoms appeared before hospitalization and were not controlled by a physician.

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AUTOINFECTION AND CONTAMINATION

In 23 of the infants, the pulmonary lesions appeared to be the first evidence of localization of the infection. The 17 others had previously had infectious foci at other sites. In the majority of these cases bacteriological investigation of these foci had not been made, but on account of their nature a staphylococcic etiology was highly probable. Among these infections, otitis media, pyoderma and subcutaneous abscesses were prominent. A peculiar feature of a few of these infectious foci is that they were clinically cured before the lungs became manifestly involved, leading one to wonder whether the pneumonia was caused by the same bacterial strain or was due to a new infection acquired in the hospital ward. Staphylococcal infections in the mothers were not recorded routinely.

It is noteworthy that 10 of the 40 infants under study were admitted to hospital for a variety of pathological states of non-infectious nature, such as malnutrition, dermatitis, icterus, hare lip, hypertrophic pyloric stenosis, and anæmia, and that acute respiratory disease appeared only during the course of treatment of these conditions. In these 10 cases it can be assumed that contamination took place in the hospital ward. The fact should be emphasized and warrants the strictest measures of hygiene.

DURATION OF ILLNESS

It was often difficult to appreciate precisely the duration of the pulmonary disease. Dyspnœa, cough, cyanosis and particularly a sudden rise in temperature were used as criteria to determine the time of onset. Death appeared after an illness that varied from a single day to 12 days, the greatest number dying after 2 to 5 days. In general, older children survived somewhat longer.

GROSS PATHOLOGY

For descriptive purposes, the pulmonary lesions may be divided into two main types: the diffuse lesions and those appearing as rather well-localized foci. No clear-cut division existed, however, between these two main forms, and intermediate aspects were a frequent finding.

Diffuse lesions.—In this type, the early lesion appeared as a bright red homogeneous area of consolidation, hypertrophying the major part of a lobe, the whole lobe, or a lobe with segments of adjacent lobes. In some cases, areas in both lungs were involved. The cut surface of these regions was rather dry and produced, on scraping, a small amount of bloody fluid. On first sight, lesions resembled red infarcts (Fig. 2). In

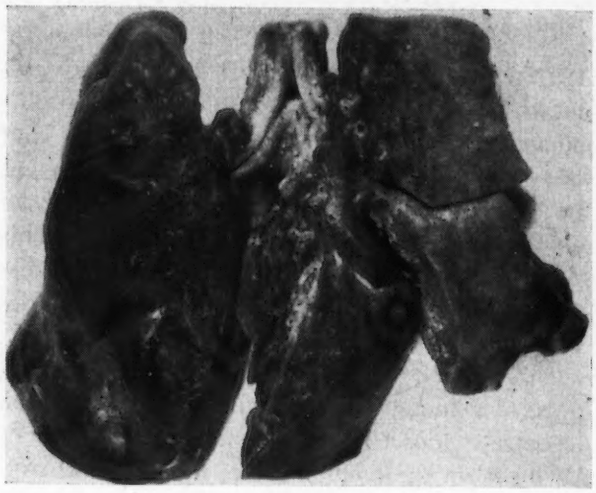

Fig. 2

older lesions multiple irregular foci of softening appeared in the consolidated areas and the cut surface yielded an abundant purulent and bloody material. In advanced cases the involved areas had the aspect of numerous and partly confluent abscesses (Fig. 3).

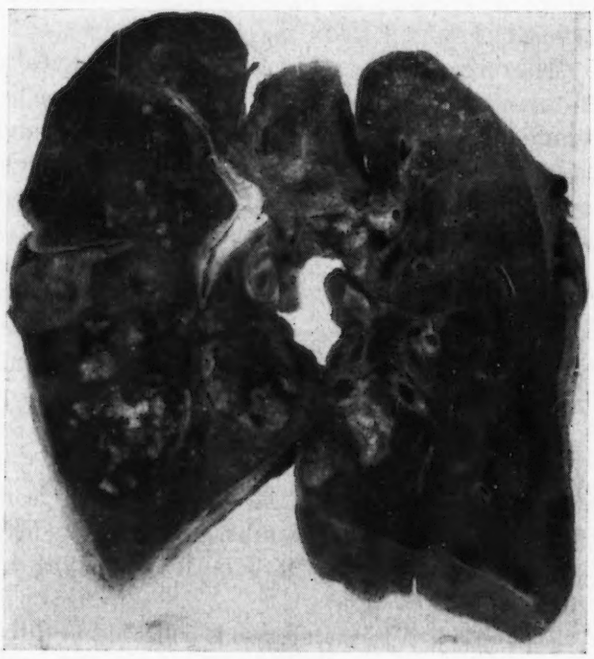

Fig. 3

Localized foci.—These were represented by rather small and sharply defined zones of hardening which seemed to undergo a rapid transformation into abscesses. These foci were

seldom unique and generally not very numerous and sometimes rather distant from one another. Most were found close to the pleura and some had ruptured into the pleural cavity, causing important exudates (Figs. 4 and 5).

In both types of lesion, one lung was mainly involved in the majority of cases, while careful examination often revealed minute lesions in the other. Of the 40 cases under review, in 17 the right side was predominantly interested, in 15 the left; in 8, both were widely and equally involved.

ing yellowish coagulated material. In two cases the liquid was slightly tinged with blood and in one case the pleural content was inadequately described. Important effusions, causing marked atelectasis, were found in most cases of the localized type of pneumonia. It seems probable that an early pleural effusion followed by pulmonary collapse had a tendency to arrest the extension of the pneumonia and consequently to produce the small abscess type of lesion (Figs. 4 and 5). In only a couple of cases did section of the lungs not reveal any abscess formation.

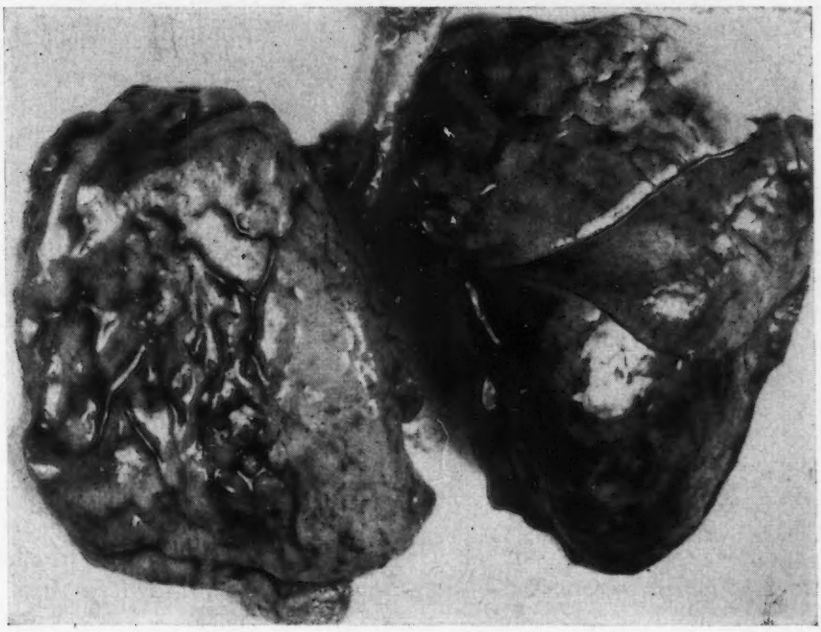

Fig. 4

When important pleural effusions were present, the non-hardened pulmonary parenchyma was collapsed. In such cases the opposite lung was most often whitish, rather large and spongy, with occasionally a few subpleural emphysematous bullæ. Emphysema was also seen in the anterior edge of the involved lung when the lesions were extensive and little pleural reaction was present.

Thirty-eight infants in our series showed pleural inflammatory reactions, most of them evidently secondary to the lung lesions. In 11 cases there were only rough yellowish membranes adhering to the pleura in the immediate vicinity of the most advanced pulmonary foci. In 27 cases there was a true pleural effusion. This varied in approximate quantity from 25 to 300 c.c. Eleven of these effusions were empyemas with or without pneumothorax, but 13 consisted in a brownish opaque liquid often contain-

These were suspected to be cases of primary pleurisy.

The incidence of pneumothorax associated with pleural lesions could not be appreciated statistically.

HISTOLOGY

In general the histological findings in the lungs were phenomena of purulent, necrotizing and hæmorrhagic inflammation, one aspect predominating from one case to another. In early diffuse lesions the parenchyma appeared to be undergoing necrosis, but was mostly infiltrated by numerous erythrocytes with a sparse polymorphonuclear exudate. In more advanced cases, necrosis of bronchial and alveolar walls was more conspicuous, and later the centre of the necrotic foci was replaced by pus. In the more localized lesions, the purulent transformation was more evident and most likely had ap-

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peared more rapidly. The histological picture in these cases could be compared to that of a carbuncle.

In the inflammatory foci, large compact clumps of Gram-positive cocci were nearly always found. The abundance of these cocci, having the characters of staphylococci, was very often remarkable and could not be entirely attributed to a post-mortem multiplication of the bacteria.

Fig. 5

Another frequent finding was the presence of necrotic and at times thrombosed blood vessels in or about the infected areas of the lungs. The thrombi often contained numerous polymorphonuclear leukocytes. Such phenomena of thromboangiitis were also prominent in early diffuse types of lesions and may explain in part their hæmorrhagic aspect. Not only was it difficult to establish the bronchogenic or hæmatogenic origin of the infection on tissue examination alone, it also was impossible to state whether these vascular changes were an extension of a necrotizing process starting from a bronchus or secondary to a septicopyæmia.

In the vicinity of the necrotic and purulent foci, persisting alveoli usually contained serous material with a few macrophagic and polymorphonuclear cells, and the capillaries were often gorged with red cells. The main bronchi were usually free, but the smaller bronchi, especially in or around the affected areas, were generally filled with polymorphonuclears. Areas of atelectasis and emphysema, when observed grossly, were also evident.

The pleura was thickened by œdema and covered with considerable fibrino-purulent membrane. When fluid effusions were present, this membrane was œdematous, partly necrotic and much less cellular. The scissuræ and interlobular walls were also thickened by œdema. In a few cases polymorphonuclears were numerous in the interlobular spaces and even abscesses were found. These seemed to be secondary to the penetration of the inflammatory process from the pleura into the interstitial tissue.

Associated Pericarditis

Nine of the 40 cases studied showed pericardial lesions. In eight of these there was no liquid formation, but only patchy, thin, greyish, rough membranes of fibrinous nature. This type of membranous pericarditis accompanied a left pleural effusion in five cases and an effusion on the right side in three cases. The secondary nature of these lesions is highly probable. In the ninth case, however, a true purulent pericarditis was present and the pleura showed only fibrino-purulent membranes in the neighbourhood of the pulmonary foci. This pericarditis was considered to be an independent lesion, and most likely had developed before the pneumonia.

Intestinal Distension

A noteworthy fact is that many of the infants had at autopsy a very distended abdominal cavity. The invariable finding on dissection was a gaseous dilatation of the whole intestine. This state of ileus, in all aspects of paralytic nature and secondary to the severe toxæmia accompanying the pleuro-pulmonary lesions, often gave clinical signs suggestive of a volvulus or an intussusception. One patient was even submitted to surgical procedures on this account.

Summary

The pathological findings in 40 autopsied cases of staphylococcal pneumonia during infancy are reported. The increasing incidence of the disease, its rapid course, its special severity in infants of one to three months of age and its

occurrence mainly during the winter months are mentioned. Stress is laid on the frequency of contamination in hospital wards. The lesions were generally more prominent in one lung than in the other and varied in aspect from diffuse hæmorrhagic zones of consolidation to well-localized abscesses. Histologically, necrosis, hæmorrhage and purulent transformation were marked features, their intensity varying from one case to another and according to the age of the lesions. Pleural reactions were a constant finding. Membranous pericarditis was noted in a few cases and toxæmia often caused a state of intestinal paralytic over-distension.

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RÉSUMÉ

Nous avons présenté une étude des principaux facteurs étiologiques et évolutifs et les constatations anatomiques dans 40 cas autopsiés de staphylococcies pulmonaires chez des nourrissons. La fréquence de cette infection semble augmenter progressivement. Elle atteint surtout les enfants âgés de 1 à 3 mois et son évolution est rapide. On la rencontre surtout durant les mois d'hiver, moins souvent au printemps et à l'automne; dans la présente série, aucun cas n'est survenu durant les mois de juillet, août et septembre. Chez plusieurs enfants, la contamination semble s'être effectuée dans les milieux hospitaliers. Les lésions anatomiques sont en général localisées uniquement ou principalement à un poumon; leur aspect varie depuis celui d'abcès multiples bien limités jusqu'à celui de condensations diffuses et hémorragiques. Les plèvres sont pratiquement toujours intéressées; elles présentent des membranes fibrino-purulentes ou des épanchements purulents ou séro-purulents. Histologiquement, les foyers inflammatoires sont hémorrhagiques, nécrotiques et purulents avec prédominance d'un aspect selon le cas et selon la durée d'évolution de la maladie. Chez quelques patients, il existait une péricardite fibrineuse. Plusieurs ont présenté un état d'iléus intestinal terminal vraisemblablement secondaire à un état toxémique.

MALIGNANT NEOPLASMS OF THE TONSIL

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CANCER OF THE TONSIL comprises slightly less than 1% of all human cancer. From 1934 to 1953, in the Hamilton Clinic of the Ontario Cancer Foundation, there were 37 cases of cancer of the tonsil, and of this group 13 were brought in for treatment in the last three years.

Although cancer of the tonsil is not a common disease, it does represent one of those miserable lesions which interfere so markedly with the function of one of the body orifices. The surprising fact is that all too frequently it is difficult to determine the original site of the growth. Usually, by the time the patient presents himself for examination, the tonsil, tonsillar pillars, adjoining tongue, and even the soft palate are involved in a fungating and ulcerating mass, and one or more enlarged lymph nodes can be felt or seen at the side of the neck.

The statistics for the 37 patients treated from 1934 to 1953 generally parallel those found in reports of larger series. *Sex incidence.*—Males predominate. In this series, there were 28 males to 9 females. *Age.*—It occurs after middle life. The majority of cases occurred between the ages of 50 and 89; only four patients were under 50 years of age. *Site of lesion.*—The majority extended beyond the tonsil when first seen. Only six were confined to the tonsil.

PATHOLOGY

There were 32 cases of epidermoid carcinoma (26 were well-differentiated squamous cell carcinoma, 1 was a transitional cell carcinoma, and 5 were lympho-epitheliomas); 2 cases of lymphosarcoma, 2 cases of sarcoma, and 1 case of plasmacytoma.

Neoplasms of the tonsil tend to arise from the squamous epithelium on the surface and lining the crypts, from the reticular connective tissue or from the lymphocytes. However, the well-differentiated extensive tumours encountered may not be primarily tonsil tumours but may spread to the tonsil secondarily from adjacent sites, such as tongue, palate, cheek, alveolus or tonsillar pillars. Some other rare types en-

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countered in other series include salivary gland tumour, leukæmia, and Hodgkin's disease.

Why do so many cases involve *cervical lymph nodes* when first seen? The lymph vessels of the tonsil are abundant, and form a close plexus around each lymph follicle. At least five lymph vessels pierce the buccopharyngeal fascia and the superior constrictor muscle to pass between the stylohyoid muscle and internal jugular vein to reach the upper deep cervical lymph nodes. Most of these vessels drain directly into the tonsillar (jugulo-digastric) node, immediately behind the angle of the jaw. In 25 of the 37 cases involved, nodes of the neck were easily demonstrable when the patient was first seen. The *duration of symptoms* was two to seven months before the start of treatment.

SIGNS AND SYMPTOMS (FIG. 1)

Pain and *throat irritation*, usually on one side, were the outstanding symptoms. The area is supplied by the 9th nerve and there may also be

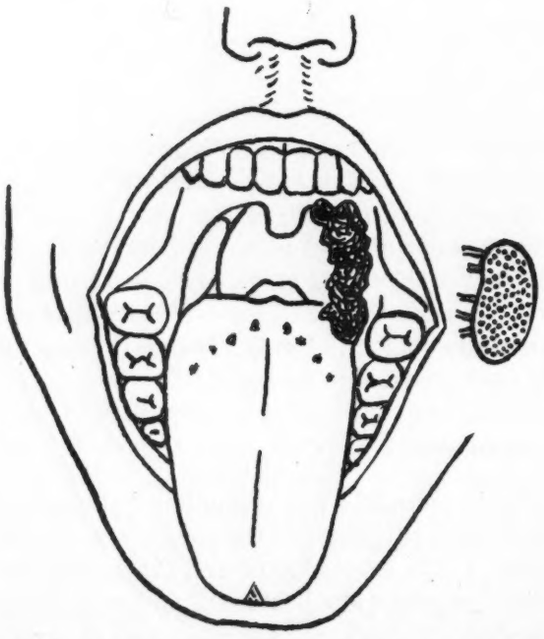

Fig. 1.—The usual appearance of cancer of the tonsil when first seen: an extensive, ulcerating tumour invading adjacent tissues and spreading early to the tonsillar lymph node.

pain transmitted by the 5th, (7th), 10th and upper three cervical nerves, depending on the extent of involvement. The region of ulceration and induration is continually irritated by movements associated with eating and talking. A secondary infection and cellulitis of the tissue around the tumour often add to the pain. Other symptoms were a *feeling of growth* or lump present in the throat; *difficulty in swallowing* with formation of mucus; discovery of a *swelling at the side of the neck*. As more tissue is involved, difficulty in talking, bleeding, dyspnœa, pain in the ear, weight loss and anorexia become evident.

Before diagnosis, 13 of the patients showed records of being treated by gargles, tooth extraction for pain, dissection of cervical node, and treatment with antibiotics for a period of one week to three months, before the primary lesion was discovered in the throat.

TREATMENT

The generally accepted treatment for the tonsil and involved cervical glands is by *external x-irradiation*. For lesions of the squamous cell type, treatment is by small-field beam-directed therapy to high dosage. For the more anaplastic types of tumour, including lymphosarcoma and lympho-epithelioma, much larger fields are used, usually opposing right and left laterals, and dosage levels are somewhat less. Daily doses are given, up to the tolerance of the tissues. In selected cases, radium implants are used in the tonsillar lesion.

The *prognosis* depends on the extent of involvement when first seen, and no form of treatment is known to salvage a satisfactory number of advanced cases. On the other hand, statistics cannot express the good palliation which can be achieved by external irradiation in a large number of advanced cases.

In rare, early cases where the tumour is confined to the tonsil, enucleation of the tonsil has been carried out. When the primary lesion is cured, it is felt by some that radical neck dissection for lymph node metastases may be considered, despite the high position of the involved nodes. But, in the main, irradiation is the treatment of choice for the primary lesion and the cervical lymph nodes.

The general condition of the patient must be kept up. Measures taken to assure adequate food and fluid intake during treatment are very important. Antibiotic drugs may be needed at times to combat local infections.

Severe pain is a problem to deal with, as pain is an early and usually persistent symptom. Local anæsthetics in liquid or powder form are helpful as well as sedatives by mouth or by injection. The tonsil region is supplied by the tonsillar

branch of the glossopharyngeal nerve. Further nerve involvement depends on the extent of the growth into adjacent tissues. Pain impulses may pass in the pharyngeal and palatine nerves by way of the spheno-palatine ganglion. The lingual branch of the mandibular nerve and branches of the vagus may also be involved. Therefore, as well as the 9th, there is great probability that the 5th, 10th (and even the 7th) and upper three cervical roots may carry some pain impulses from this region. For intractable pain, several procedures have been used to alter the pain stimulus, with varying success: alcohol block of the region of the spheno-palatine ganglion, section of the lingual or glossopharyngeal nerves, prefrontal lobotomy and tractotomy of 5th, 9th, 10th and upper three cervical nerves.

Response to therapy is generally disappointing except in lympho-epithelioma and early localized lymphosarcoma, which represent a minority of the cases.

In this series 23 died within 2 years; one did not return and is presumed dead; 3 lived 3½ years; one lived 7 years; 2 lived 12 years, and 7 are now living. Of those now living, 5 started treatment within the last 2 years and all but one are well and without recurrence. One at 7 years and one at 10 years are well without recurrence.

This presents a somewhat gloomy picture, but the opportunity to head off this disease early may arise if the following are kept in mind: (1) In a case of unilateral enlargement of the tonsils which is inflamed and does not respond to local treatment within a few days, biopsy should be done so that an early diagnosis of malignancy can be established and therapy instituted. (2) In a male patient over 50 years of age who complains of throat irritation or pain on swallowing, a good examination should be carried out using a light, a tongue depressor, a finger cot for digital examination and perhaps local anæsthesia if the gag reflex is vigorous. One must think of looking for *asymmetry, ulceration* or *induration*, as these are the early changes.

CONCLUSION

An outline of the findings in 37 cases of cancer of the tonsil is shown to bear out findings in other larger reported series.

The surprising length of time that patients endure symptoms in the throat before seeking help is remarkable, as well as the advanced and readily recognizable stage of these cases before a biopsy is taken and treatment instituted. The cure rate is generally disappointing. The method of treatment is outlined.

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Case Reports

THROMBOPHLEBITIS MIGRANS AND THROMBOPHLEBITIS GENERALLY

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ON THE BASIS of my personal experience as a victim of the migrating type of phlebitis, I wish to present a new approach to the entire problem of thrombophlebitis (as distinct from phlebothrombosis).

I found myself bedridden with an inexorably advancing disease, resistant to all therapy and poorly documented in the literature. The instinct of self-preservation stimulated the processes of thought and led to my developing a working hypothesis based on clinical reasoning and effective in practice.

Inflammation as violent as in my case suggested infection, which was however ruled out by the normal pulse, temperature, erythrocyte sedimentation rate, and white cell picture, absence of regional lymphadenitis, and lack of response to antibiotics. The other possible cause was trauma. Even slight trauma seemed to initiate the process, so it would seem that there must be

some underlying constitutional defect in the tissues attacked. This thought led to the conviction that phlebitis was but a local manifestation of a constitutional disease. The violent reaction in the vein and surrounding cellular tissue still had to be explained. Clearly some irritant agent was at work, and since infection was excluded the irritant must be a chemical one, either arising deep in the tissues or reaching them in the bloodstream.

This concept suggested a metabolic disturbance, on the analogy of local phenomena in diabetes, which however was not present. A disturbance of protein metabolism seemed likely, since the end-products of such metabolism, violent tissue irritants, are known to cause severe inflammation. We also know from our studies of gout that uric acid crystals deposited in the tissues are just such inflammatory agents.

Recent electro-chemical research has shown the existence of a large "miscible pool" of uric acid, widely distributed throughout all the tissues of the body of the gouty individual—tendon sheaths, synovial membrane, bursæ, cartilage, bone, ligaments, blood vessels, and the connective tissues generally. These reports were not available to us at the time.

The popular conception of gout seems to have focused attention on an excruciatingly painful inflammation of the big toe, almost to the exclusion of all other affected tissues throughout the body. I would go so far as to say that *most other manifestations of gout are seldom diagnosed as such.*

At this point in the development of our theme, extensive blood chemical tests were done. Every single result was normal, except for a consistent slight elevation of blood uric acid. (Blood uric acid tests are generally unsatisfactory and inconclusive and can be near normal while electrochemical tests will show the presence of an abnormally large "miscible pool" in the tissues.) Any attempt to attach the same significance to the actual mathematical figure as we do to the blood sugar level in diabetes would be unrealistic. Any attempt to gauge the size of the "miscible pool" by means of this test would be erroneous and misleading.

I recalled the appearance at intervals over the preceding years of small exquisitely painful areas of inflammation in the soft tissues over the malleolus of the ankle, the epicondyle of the humerus, the ligaments at the knee, the dorsum of the wrist, the pulp of the fingers, and the long head of the biceps. For many years before the outbreak of phlebitis, increasing pain, stiffness and disability related to all the joints of the body was in evidence. Attacks of "acute lumbago", "muscle cramps", "muscular rheumatism", "wry neck", etc., increased in frequency and severity, and in retrospect a diagnosis of "gouty diathesis" seemed reasonable. Water retention was also a definite feature. About three months after the onset of the phlebitis I developed an acutely painful, swollen and inflamed little toe, which was considered "gouty" by my colleagues.

Was the phlebitis also a local manifestation of the gouty diathesis? This suggestion met with some scepticism, but intensive anti-gout therapy was nevertheless instituted. The response was nothing short of miraculous. Pain subsided within 24 hours. Inflammation was receding within 48 hours. The entire course of the disease was suddenly reversed. Recovery was rapid, complete and seemingly permanent. I have been back at work for three years now, with no recurrence of the phlebitis.

Supportive evidence.—While I was still in hospital, one of my colleagues turned over to me the management of a case of thrombophlebitis migrans of nine years' duration. Nearly all the veins in this patient's body had been involved at one time or another. Most of the deep circulation was obliterated and an extensive network of large collateral veins had developed, especially over the abdomen. The current attack of phlebitis was in one of these. Both legs were swollen with hard brawny œdema and pitted with intractable and painful ulcers which would not heal. Multiple pulmonary emboli had brought him close to a fatal termination in a previous attack involving the veins of the left arm. His blood uric acid was elevated.

Intensive anti-gout therapy was begun and has been continued to date. Response to therapy was even more spectacular in his case, and now, three years later, he is still entirely free of phlebitis. The œdema in the legs has substantially subsided. The ulcers healed rapidly and have remained healed. He is completely rehabilitated and working full-time. He, like myself, will continue the rigid anti-gout regimen throughout life, just as a diabetic must continue diet and insulin throughout life.

Now, what of postoperative phlebitis and the ordinary superficial thrombophlebitis seen in general practice? In the past two years, not one single case of thrombophlebitis which has come under our jurisdiction has failed to respond rapidly, dramatically, and completely to this therapy. Pain is reduced within 24 hours, inflammation begins to subside within 48 hours, and progress to recovery is a matter of days, rather than weeks or months. Two cases with showers of pulmonary emboli were out of danger much more rapidly than with any other form of therapy we have ever tried.

I am inclined to condemn the use of anticoagulants in the light of this concept, and its subsequent support by clinical experience. With this therapy, the process begins to resolve in a shorter period of time than it would take for anticoagulants to have any appreciable effect. Anticoagulants are dangerous, and do nothing to cure the phlebitis itself.

Ligation of veins seems also to be contraindicated on the basis of adding surgical trauma at a time when the stage is set for the development of more phlebitis, and in the fear that it would only encourage what it seeks to prevent.

Will this regimen continue to work in all cases? Will its usefulness be limited to a specific group of cases, while others fail to respond? Time alone will tell. The blood uric acid level was elevated in some of our cases, normal in others. The treatment seems as effective in the one as in the other.

Many questions I cannot answer. I simply throw these results out as a challenge to someone with the time and the laboratory facilities to investigate as a problem worthy of research. Since no man in his lifetime sees enough of these cases to be able to impress with the weight of statistics, I make no apology for the small number of cases. It is to be hoped that interested readers will add to the statistics. Any evidence which tends to prove or disprove the validity of my theory will be welcome.

I have waited three years from the date of cessation of symptoms (in myself and my patient) of the migrating type of thrombophlebitis before venturing into print. In the meantime several of my colleagues and I have observed the effectiveness of this treatment in all other types of thrombophlebitis under our care. Is it pure coincidence that therapy specific for gout should also seem specific for thrombophlebitis?

Many questions remain unanswered. Is this primarily an endocrine dysfunction? The profound effect of the pituitary on the rate of urinary excretion, as evidenced by the upset of this function in diabetes insipidus, at least suggests that this gland may be suspect. Recent publications tend to show that cortisone is effective in both acute gout and acute thrombophlebitis. There are those who believe that aspirin stimulates the production of adrenal cortical hormones. Are the adrenals involved? There is both increased production and impaired elimination of uric acid in gout. Kidney function sooner or later is impaired. Is this an end result or a primary cause? Is this dependent upon pituitary or adrenal dysfunction? These are but a few of the many questions.

Nowhere in the recorded literature had I come across any suggestion of a relationship between gout and thrombophlebitis, but there is a short paragraph in the British Encyclopædia of Medicine which mentions the startling fact that Paget in his day described a "gouty phlebitis" which in all particulars paralleled my own case. For some reason, this has been entirely ignored ever since, yet none of us questions Paget's remarkable powers of astute clinical observation.

I will welcome reports from colleagues who try out this treatment, summarized below, with an assessment of its value in their own practices.

TREATMENT

Immediate.—Liquids only and non-protein soft solids; 60 grains of aspirin daily; 6 to 8 tablets of colchicine (grains 1/100) daily, to be reduced to 3 or 4 tablets daily if too much intestinal misery ensues. (Colchicine alone is not enough; aspirin alone is better; the combination of the two is most effective.) Complete and absolute bed rest, on the ground that exercise causes increased production of uric acid and can precipitate acute attacks of gout in a gouty individual. (The week-end round of golf in a person of sedentary habits is a case in point.)

Long-term.—Avoidance of strenuous exercise. Early to bed, late to rise. Strict diet, low in protein generally with particular avoidance of foods high in purine content. Benemid (2 tablets daily) as maintenance therapy. Periodic aban-

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donment of this for stretches of two or three weeks, during which intensive colchicine and salicylate therapy is given "to wash the uric acid out of the tissues". Salicylates and Benemid should not be used together.

Salicylates do reduce the "miscible pool" of uric acid in the tissues. This pool can be of such magnitude that it may take months to bring about an appreciable reduction. It is relatively insoluble, yet continually being added to or subtracted from in the exacerbations and remissions of the disease. Salicylates seem most effective in practice. I have found Benemid satisfactory for maintenance therapy, but drop it in favour of colchicine and salicylates at the first premonitory signs of the general gouty diathesis threatening to break out into an acute localized lesion. I have had no phlebitis since treatment was first initiated, but have had one or two acute lesions in and around the soft tissues of elbow, wrist and knees. These fade out in 24-48 hours on intensive aspirin and colchicine therapy, and resolve in about 10 days. This long-term treatment is essential; discontinuance of therapy as soon as the phlebitis subsides will only bring the treatment into disrepute.

It is not difficult to diagnose an advanced and hopeless case of gout when the classical signs which develop late in the course of the disease are apparent. What seems to be less obvious, and certainly less readily accepted by the profession, is that every gradation of severity occurs from the mildest borderline case to the most advanced. Most of them go undiagnosed. Perhaps recently reported electro-chemical tests, using radioactive nitrogen, will open the way to a better understanding of this disease if they come into common and practical use in hospital laboratories.

In the meantime, it must be realized that the localized pathognomonic lesion, which is simply "a straw in the wind", may be an infrequent occurrence. Until it can be proved otherwise, it would seem reasonable to treat thrombophlebitis as a localized manifestation of gout, on the grounds that *adequate* anti-gout therapy has been so convincingly effective to date.

I acknowledge my indebtedness to Dr. E. M. Wilder and Dr. A. C. DesBrisay for medical care at the time, and for the discussions which helped to formulate this concept.

RECRUDESCENT TYPHUS (BRILL'S DISEASE)

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IN 1898 BRILL[1] observed a disease resembling typhus fever in immigrants from Europe to the United States. During the next few years similar cases were seen in many parts of America. Zinsser in 1934[2] reviewed 538 cases of Brill's disease and advanced the hypothesis that this disease was a recurrent form of typhus fever, the primary attack occurring in an area where epidemic typhus was prevalent. Zinsser postulated a latent period during which the typhus organisms persisted in the tissues, the factor responsible for a recurrence of the active disease being unknown.

The clinical picture of recrudescent typhus is similar to that of epidemic typhus, although the course of the disease is generally less severe. The rash and the serum agglutinins for *Proteus* OX 19 associated with epidemic typhus are both usually absent in recrudescent typhus.

Mrs. T.M., 51, Polish Ukrainian, was admitted to the Winnipeg General Hospital on November 6, 1953, with the following history.

During the summer of 1953 she complained of a cough, excessive lacrimation and a choking feeling in her throat. Her physician made a tentative diagnosis of goitre, and iodine therapy was instituted. The patient felt reasonably well until October 1, 1953, when there was an abrupt onset of fever and headache. The headache was situated at the vertex and was sharp and continuous; there was no frontal or occipital aching. On November 2 and 3 she vomited and had occasional chills. On November 4 she was given an injection of 400,000 units of penicillin. This treatment had no effect on the fever, but there were no further chills. On admission to the hospital the patient complained of feeling feverish and of an extremely severe headache.

In 1928 an ovarian cyst and the appendix had been removed. In 1951 a hysterectomy had been performed. The patient had had a previous episode of dermatitis.

Physical examination.—The patient did not appear unduly ill although her temperature was 104° F., pulse rate 120 and respirations 20. She was complaining bitterly of the headache. There was no neck rigidity. With the exception of a palpable isthmus of her thyroid, the remainder of the physical examination was essentially negative. There was no rash and the spleen was not palpable at any time during the illness.

On November 6, the leukocyte count was 9,000/c.mm., with 80% mature neutrophils, 4% young neutrophils, 13% lymphocytes, 1% monocytes and 2% degenerated cells. Hæmoglobin value was 90%; red cell count 4.8 million/c.mm. E.S.R. (Westergren) was 67 mm. in one hour. A lumbar puncture was performed with

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TABLE I.

AGGLUTINATION REACTIONS

Date of blood collection	*Nov. 9, 1953*	*Nov. 23, 1953*	*Dec. 22, 1953*	*Nov. 22, 1954*
Proteus OX 19 agglutination	1/320			1/20
Complement-fixation test:				
Epidemic and murine combined typhus antigen		1/256	1/64	1/8
Epidemic typhus antigen		1/512	1/256	1/16
Murine typhus antigen		1/32		0
Blood samples were later sent to Dr. Michael Sigel, Communicable Disease Center, Montgomery, Alabama, for complement fixation tests for confirmation:				
Epidemic typhus antigen	1/1024	1/1024		
Murine typhus antigen	1/256	1/64 or 1/128		

an initial pressure of 240 mm. of water and a final pressure of 200 mm. Five c.c. of clear fluid was removed which had a total protein content of 10 mg. %: the fluid contained 17 leukocytes and 5 lymphocytes per c.mm. Colloidal gold test was negative. Urinalysis was normal. Two blood cultures were reported negative. The roentgenogram of her chest was normal. Blood agglutinations are reported in Table I. On November 9 the leukocyte count had increased to 16,000, with a differential count of 60% mature neutrophils and 18% young neutrophils showing toxic granulation, 13% lymphocytes, 8% monocytes and 1% degenerated cells. A second urinalysis revealed a trace of albumin.

In the absence of a definitive diagnosis, no antibiotics were given. On November 6, 7 and 8, the patient's temperature rose from 104 to 105° F., subsiding to 99° F. on November 9; the patient then remained afebrile until she was discharged from the hospital on November 14. On November 23, when a provisional diagnosis of recrudescent typhus had been made, the patient returned for further questioning. She stated that in 1918 or 1919, when in the Polish Ukraine, she had suffered an attack of the "flu"; the members of her family were also sick and the woman nursed them in spite of her own disability. Later she became dangerously ill for a period of three weeks and had been informed that she was near death during this period. In the course of this illness all the hair of her head fell out. The patient emigrated to Canada in 1921 and until the present attack had suffered no illness with symptoms resembling those of 1918-1919.

DISCUSSION

A rickettsial infection was suspected in this case when the *Proteus* OX 19 agglutination test was positive; this finding is unusual in recrudescent typhus. The complement-fixing antibodies had apparently reached a peak in the serum by the time the first blood sample was collected, 10 days after the onset of illness. As shown in this case, specific serum antibodies increase much more rapidly in recrudescent typhus than in the primary attack of epidemic typhus. The third sample, collected 52 days after the onset of the disease, showed a slight decline in antibody content compared to the second one. The fourth sample, collected approximately one year after the illness, showed a marked decrease in the antibody titre.

The complement fixation test with epidemic typhus antigen showed the highest titre of antibodies; using the murine typhus antigen, a low but definite titre of antibodies was obtained. These results illustrate the cross-reaction that other workers have observed between these two antigens.[3]

A case of recrudescent typhus in Canada is primarily important as a possible focus of infection from which an epidemic of classic epidemic typhus could originate. This potential hazard to the health of the public appears to be the most significant aspect of cases of recrudescent typhus in this country. In view of the large number of immigrants from Central Europe, where epidemic typhus has been rampant, it is surprising that more cases of the disease have not been reported in Canada. The possibility exists that some undiagnosed fevers in immigrants may be recrudescent typhus.

Epidemic typhus flourishes in countries where a cold climate and poor living conditions predispose to an increase of louse infestation. Certainly the winter season in Manitoba is cold and, although living conditions are generally good, every large city possesses a section where overcrowding supplies the necessary requirements for the spread of epidemic typhus.

SUMMARY

1. A case of recrudescent typhus has been reported.

2. The diagnostic procedures are reviewed.

We wish to express our appreciation to Dr. G. P. Fahrni for permission to publish this case.

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ACUTE INTESTINAL OBSTRUCTION IN A NEWBORN INFANT

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COMPLETE OBSTRUCTION of the intestine is not common in the first few weeks of life and is usually due to a congenital failure of development of a segment of the gastrointestinal tract, or to an abnormality of pancreatic exocrine function causing viscid meconium. Intussusception in the neonatal period is extremely rare. In the literature reviewed no case has been found where obstruction occurring in the first few days of life has been due to volvulus of the intestine round a band running from Meckel's diverticulum to the umbilicus. Although this case presented the typical features of acute obstruction, it is described because of the apparent rarity of the cause.

Baby M. was delivered spontaneously after an uneventful labour lasting about three and a half hours, the only abnormality being rotation of the head through 180° from the left occipito posterior position to the right occipito anterior. Self-administered trichlorethylene was used during the second stage with contractions. No other drugs were given. The child was a healthy male weighing nine pounds, with no evidence of congenital abnormality, and cried lustily with the delivery of the body. A small quantity of mucus was sucked from the pharynx. The third stage of labour was normal.

During the first 40 hours of life the baby behaved normally, except for a small regurgitation at 28 hours. Urine was passed twice and there were normal meconium stools at the 6th, 18th and 20th hours. A small amount of meconium was passed at 51 hours. The baby had been sucking well at the breast and taking small amounts of 5% glucose water, but fed poorly at 44 and 48 hours. At 52 hours, just after breast feeding, he vomited a large amount of greenish fluid which hit the wall a foot away. Four hours later there was another large emesis, but it was not projectile.

At this time (56 hours), although distension was clinically slight, radiographs of the abdomen showed a large amount of gas. In spite of frequent aspiration from the stomach of small amounts of yellowish mucus and fluid, the distension persisted, dehydration became apparent, and, at 61 hours, subcutaneous administration of fluid was begun. Crystalline penicillin 50,000 units and vitamin K 5 mg. were given intramuscularly.

When the infant was 63 hours old the abdomen was opened through a right lower paramedian incision four inches long, under oropharyngeal ether and oxygen anæsthesia. There was a small amount of clear yellowish fluid. The upper two-thirds of the small intestine was greatly distended and the lower third completely collapsed. A Meckel's diverticulum about 0.5 cm. long with a broad base was attached by a fibrous band to the umbilicus, and just distal to this the ileum was twisted round the band with complete occlusion of the lumen. The band was ligated at its attachment to the diverticulum and divided. Immediately the distal ileum began to fill, and the calibre of the small bowel was uniform before the peritoneum had been closed. No further operation was done and the abdomen was closed in layers.

Postoperatively the infant was placed in an incubator in a 60% oxygen atmosphere. The main features of the postoperative course were a temporary rise in temperature to 105.4° F., the institution of oral feeding 12 hours after the operation, and the return to parenteral feeding for the period from 36 to 60 hours postoperatively because the oral route was poorly tolerated. Thereafter the infant made an uneventful recovery and is in excellent health at the age of 15 months.

The mother has three older children, all healthy. Two weeks later a first cousin of this child was born with a blind rectal pouch separated from the sigmoid colon by a membrane.

Special Article

PENSIONS AND TAX RELIEF FOR PHYSICIANS

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"IT HAS TO BE REMEMBERED that the law of income tax is not one of the laws of nature. It is purely man made and the principles governing the imposition of the tax therefore depend on the will of the creator."

This somewhat salutory statement is taken from the Millar Tucker Report to the British House of Commons "On the Taxation Treatment of Provisions for Retirement". It is used in referring to the changes needed in the income tax law as it applies to self-employed persons, as are most physicians. It is they, the self-savers, who suffer from this "discrimination" in our present Canadian Income Tax Act, as opposed to the pension-savers who, because they work for a group or an industry, are allowed postponement of income tax on a portion of their income, if they are eligible for membership in approved pension plans.

A strenuous and, if necessary, a more organized effort should be made by the Canadian medical profession to influence "the will of the creator". It is 10 years since this matter was first presented to the Ives Commission of our Canadian Parliament and no progress has so far been made. In

1953, during the reading of the bill to amend the Income Tax Act, the former Minister of Finance outlined the problems involved "in the hope that it would be subjected to close critical analysis not only by members of the House of Commons, but also by those who are interested in the problem". The members of the House have shown deep interest in this pension problem themselves, to the extent of setting up a highly satisfactory scheme of retirement income for members of the House of Commons.

There are good historical precedents for tax relief for retirement income of the self-employed, and present-day recognition of the need for such plans is seen in the legislation of the U.S.A. and Britain.

When Pitt introduced the first real Income Tax Bill in 1799, he allowed deductions for life assurance, and although this provision was dropped in 1842, it was reinstated in 1853 by Gladstone, with the avowed purpose of benefiting those with precarious incomes, that is, professional men and others, dependent on their own exertions. Such relief is still afforded the British taxpayer. Even though this may not be too great a relief, it is nevertheless a recognition of and an attempt to meet a need.

The Tucker Committee in its report was satisfied that it was impossible to maintain an adequate standard of professional living and at the same time provide for retirement income under the present system of high taxation. For self-employed Canadian physicians, and this represents 90% of Canadian doctors, the same situation exists.

The physician suffers with all the self-employed under the inequities of the present system, but he suffers more because he requires the longest training amongst the professional groups. His earnings are concentrated in a shorter period of time, and during these peak years he is often taxed in a higher income bracket than if he had had his income spread out over a longer tax period.

The effect this situation is having on the profession is noteworthy, and even the lay press is interested in the problem.

This is seen in an editorial in the *Saturday Evening Post* of April 24, 1954, entitled "Why Not Encourage the Self-Employed to Build Their Own Retirement Fund". The *Post* shows what is happening in the U.S.A., and the Canadian situation is not vastly different. "In the Philadelphia area recently," it instances, "a physician enjoying a large and presumably profitable private practice, retired and took a job in a Government hospital. He gave as his reason for doing so the fact that he had been unable to educate his children and at the same time provide for eventual retirement. He felt himself compelled to become an employed person in order to receive the benefits of a pension fund."

Instances of the "old doctor" who has had to hang on to his practice because he has nothing on which to retire are too well known to all of us. Or the middle-aged physician who is ill and who has served his hospital, his patients and his community well, and yet can't take it easy or retire, as he was not able even at his peak years to save after paying his income tax.

Where are the young graduates who could be our much-sought-after general practitioners or self-employed family doctors? They are to be found in the clinic groups or among the company doctors, where among other things they will have a certain security which appeals, and a guaranteed retirement income paid for by income tax relief allowed to them because they are *not* self-employed.

The *Journal of the American Medical Association* of July 30, 1955, states that the Ways and Means Committee of the House of Representatives has recommended the passage of a bill to authorize the deferment of income taxes on money put into annuities. The objective is to give physicians and other self-employed persons the same tax advantages that are enjoyed by employees of corporations. Under the Bill a taxpayer could set aside 10% of his income for tax deferred annuities with $5,000 annual limitation and a lifetime limitation of $100,000.

The American Medical Association has worked hard for this legislation, and its success should spur the Canadian Medical Association to greater efforts at Ottawa on behalf of Canadian physicians.

There *is* Canadian interest in this problem. The *Financial Post* of July 30 in an article "Will Self-Employed Canadians Get Tax Relief on Pensions?" discusses the American and British situation and shows the Millar Tucker recommendations to be more generous for the self-employed than the American.

The Financial Post goes on to outline the Canadian position and states, "In Canada no tax postponement scheme has any official blessing yet. Any scheme that might be considered would have to be related to the provisions for employees' approved pension plans. Under such schemes the maximum annual tax-free contribution is fixed at $1,500. For the self-employed this should be doubled to $3,000, because there is no corresponding employers' contribution."

"There is nothing so certain as death and taxes." The medical profession has contributed in no small degree to the delaying of death, and thus has increased the need for pensions. Now the Canadian profession and particularly the Canadian Medical Association is faced with the second problem, namely, the *delaying* or deferring of taxes, so that the self-employed physician may provide more adequately for "dying old."

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Editorials

NATURAL CHILDBIRTH

During the last two decades there has been a movement, led by Grantly Dick Read in England and by Velvovski in Russia, in favour of more active preparation for childbirth, as a revolt against the increasing mechanization of the process. It would appear, from the statements of Paris obstetricians who have visited the Russian school, that the methods of both these protagonists are similar, although the Russians have built up a theoretical background for their activities based on the school of Pavlov and prefer to regard their prenatal work as a conditioning for labour rather than a simple physical and mental preparation. Two papers published recently, one from France and the other from the Netherlands, give a fairly clear picture of the method of psychoprophylaxis in pregnancy. Lepage and Langevin-Droguet (*Presse méd.*, 63: 549, 1955) are careful to point out that the terms "natural childbirth" and "painless childbirth" are misnomers, used in connection with this type of preparation for labour. Some 90% of labours are spontaneous, whatever the method of preparation, and therefore "natural", and physical and mental preparation for labour cannot abolish pain, which is a fundamental physiological attribute of uterine contraction. All that prenatal preparation can do is to make the pain tolerable for a certain proportion of women, abolish the accompanying anxiety and apprehension, and put the woman in the best possible condition for her work. This is not a new aim. Van Eps (*Lancet*, 2: 112, 1955) quotes the writings of a Dutch obstetrician, Wigand, who in his 1822 textbook devoted several pages to the psychological aspects of labour. Read and Velvovski have simply organized a system of preparation and have contrived to popularize it, as well as put it on a physiological basis. The chief result of such preparation for labour may be to enable the parturient woman to retain her free-will during her accouchement.

Both Lepage and van Eps report series of cases in which the method has been used in controlled conditions. Van Eps mentions that he has had the advantage of working with a population not habituated to anæsthesia in labour, and in a culture where the parturient woman is expected to behave in a disciplined and decorous manner. He had two series collected since 1949 in Amsterdam; 335 women were carefully prepared for labour by instruction, exercises, breathing and relaxation training, while another 335 were not prepared at all. The basis of selection is not mentioned, whereas Lepage, who reports results also collected since 1949 on 40 prepared primiparæ and 40 unprepared primiparæ, gives a basis for selection. He selects primiparæ, anxious and frightened women, scoliotics, kyphotics, women with pelvic contraction, obese women and those who have previously undergone a long or difficult labour. He considers that the method is contraindicated in tuberculosis, cardiac disease, albuminuria, habitual abortion, pelvic inflammatory disease, thyrotoxicosis or diabetes.

Lepage lays great stress on the need for starting preparation early, i.e. at the third or fourth month. For example, among women who had had 24 weekly sessions of one hour—including 20 minutes of gymnastics, 25 minutes of relaxation and a preliminary session of 10 minutes' massage during which the woman receives a certain amount of psychotherapy—23 were calm during labour and only one was agitated while 21 had good expulsive efforts. Of those prepared by only 6-24 sessions, seven were calm and four agitated and the proportion having good expulsive efforts was lowered. At the sessions the participants should acquire a sensation of internal calm as well as of physical wellbeing. Twice a month the women are given instruction on the anatomy and physiology of labour, and efforts are made to prevent the sessions from being a bore. In this way the woman comes to hospital in labour and feels at home at once.

In describing his results Lepage points out the difficulty of assessment, since there is no means of knowing how the women would have fared without preparation. To question them later about their experience is useless, since the memory of a bad confinement is often quickly lost in the joy of motherhood. Taking objective criteria, he finds that preparation does not shorten the total length of labour; with this van Eps agrees. It does shorten the second stage (in the Paris series 33 women in the prepared series had a second stage of less than 30 minutes as against only 24 in the unprepared); van Eps finds only a slight shortening. Rigidity of the perineum was much less common in prepared women (10 out of 40 as against 19 out of 40) and tears were also less frequent (6 against 11). None of the prepared group needed forceps; 5 of the others did. The Dutch clinician reports that the tear and episiotomy rates were only insignificantly lowered by preparation (48.6% as against 50.6% in primiparæ). Lepage mentions the great change in the atmosphere of the labour room when the woman has been prepared.

Van Eps classifies his results according to the method previously used elsewhere as *very satisfactory, satisfactory, fair* and *poor*. By preparation the proportion of *poor* results fell from 17% to 5%. This last figure agrees pretty closely with those from French, Russian and American clinics. The *very satisfactory* figures also agree well (44-50%).

Lastly, van Eps studied the problem of determining which women would be most likely to benefit from preparation, by carefully investigating their emotions and reactions during pregnancy and labour and their social and personal history. In several cases the difference in behaviour of a woman during the first and second stages could be related to her personality. The active type found the first stage more difficult, while the passive type regarded the expulsive stage as harder. Behaviour during labour could be fairly well foretold. In the series there were 11 well-balanced and well-adjusted women, stable, devoted and self-confident. In all these cases results were satisfactory. On the other hand, of 13 neurotic, egotistical, unstable women only 2 had a satisfactory result. The next problem is what to do with the 5% who will have poor results. According to van Eps individual psychotherapy may be needed but the outlook will probably remain poor.

Editorial Comments

YOUR RETIREMENT INCOME

Nearly everyone agrees that the provision of adequate retirement funds is a desirable objective in this age when social security is a concept which engages the attention of all of us. In Canada, our Government has encouraged and assisted us by providing modest pensions for all at age 70 and in certain circumstances at age 65, by permitting the thrifty to buy Government Annuities at advantageous rates and by providing for some of our fellow citizens who are members of approved pension plans, the incentive of income tax relief on their personal contributions to the fund. Dr. Quintin's article on page 562 of this issue points out that the self-employed taxpayer has not been afforded the same practical incentive to make provision for his own retirement.

In considering this apparent inequity it should be remembered that the advantage enjoyed by members of approved pension plans is one of tax deferment. They are permitted to deduct from taxable income their personal contributions to the pension fund (monthly retained as payroll deductions) in the year in which they are made but they pay tax on the annuity or other pension income in the year in which it is received. In so doing they pay taxes not only on their own investment in the fund but also on their employer's contribution. The self-employed currently may make retirement provision by the purchase of annuities or life insurance in one of its many forms. He pays taxes on his premiums but pays no tax (or practically no tax) on the retirement income when he receives it. The self-saver lacks the neat organization of an employed group as well as an employer and it would appear that this administrative handicap has been a major obstacle to the recognition of our claim for equal treatment. Membership in a recognizable group, such as the Canadian Medical Association, has not yet been accepted as a substitute for the employee-employer relationship.

Reference has been made to the Tucker Committee in the United Kingdom and to action of the Ways and Means Committee of the House of Representatives in the United States. Both of these bodies have recommended changes in the law or in its administration to provide tax deferment for pensions of the self-employed, but in neither instance has the necessary legislation been effected. The Canadian Medical Association has made repeated representations both individually and in co-operation with the other professional groups to our own Government. We have stood aside while the lawyers and the chartered accountants have exercised their persuasive powers. We have suggested methods of administration varying from one resembling the

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compulsory saving procedure of wartime taxation, to an Association annuity plan or a trusteed pension plan which might involve us in the insurance field to a greater degree than many members would like. The matter has been debated in Parliament and the justice of our contention has received much support. Dr. Quintin's clarion call for further efforts to influence "the will of the creator" will be heeded by the Income Tax Committee in their endeavour to correct an anomaly.

NEUROMUSCULAR DISORDERS

Some disease conditions seem to have attracted the attention of the public and the scientist ever since the dawn of medical research; others—equally important in terms of human distress—find brief mention in the closing sections of medical textbooks and remain an enigma to this day. The neuromuscular disorders belong to this latter group; muscle physiology and pathology are still poorly understood, and attract comparatively few research workers.

We welcome the paper by Dr. Kanaar on page 532 of this issue, in which he explains the work of the Muscular Dystrophy Associations of America and of Canada, and tells us something about the first Muscular Dystrophy Clinic in Canada. There are now quite a number of voluntary associations for the study of diseases such as diabetes, tuberculosis and multiple sclerosis, and the question may be asked whether comparatively rare disorders like the muscular dystrophies merit an addition to the list. We believe that they do, for two reasons: (1) The main point in the Canadian Association's programme is stimulation of research into all neuromuscular disorders (not only the dystrophies proper but also myasthenia gravis, etc.), a hitherto neglected field. It is the intention of the Association to provide research grants previously not available, and to give fellowships to promising workers. In doing this, the Association is going right down to bedrock, encouraging not only clinical research but also basic research on muscle. (2) The C.M.D.A. is giving hope and help to the unfortunate sufferers from these so far incurable conditions, raising their morale, helping to rehabilitate them, and incidentally collecting them together in numbers suitable for statistically controlled study.

The President of the Canadian Muscular Dystrophy Association, now entering a second year of vigorous development, is Mr. Arthur Minden, a Toronto lawyer; the Vice-President is Dr. David Green of Toronto. Interested persons are asked to direct inquiries to the Association at Room 507, 137 Wellington Street West, Toronto.

ENDEMIC GOITRE IN SOUTH AFRICA

The Department of Nutrition, Union of South Africa, has recently published a report on endemic goitre.[8] It is an account of the work of the South African Goitre Research Committee. The report is in two sections, one being a summary of current knowledge and the other being an account of an extensive survey of the condition amongst various racial groups in different parts of the country.

From this report it is clear that primary iodine deficiency in soil and water is not the only factor in the etiology of endemic goitre. The authors rightly discuss the possible influence of bacterial pollution of water, originally mentioned by Sir Robert McCarrison in India,[6] of other halogens, calcium, iron and arsenic and deficiency of copper. Especially interesting is the review of the chemically complex goitrogens and thyrostatic drugs ranging from the thiocyanates to resorcinol, thiopental, "Antabuse", the thiouracils and possibly even sulfaguanidine and the barbiturates. Most of the latter group would not be generally operative in the South African scene, but they may be of importance in individual cases.

In most parts of the country where goitre is found, the cause is ascribed to primary iodine deficiency in the soil and water and foods grown in the particular areas. These differ from the high rainfall coastal belt, the George and Knysna districts, near Port Elizabeth, in which soil and water lack not only iodine, but also calcium, phosphorus and magnesium, to parts of the Transvaal Highveld, where water is hard in most places. Lacking in iodine, mountainous Basutoland is an endemic goitre area, as is also the swampy Caprivi Strip, which is a part of South West Africa bordered by Angola, Northern Rhodesia, Southern Rhodesia and the Bechuanaland Protectorate. In the Caprivi Strip soils are fertile, food supplies are satisfactory and certain natives eat a great deal of river fish. The cattle in this area are in good condition and show no signs of goitre, a rather puzzling situation. This is ascribed to the fact that the grasses eaten by stock in human endemic goitre areas contain relatively more iodine than the vegetables, cereals and fruit grown in those areas. Why this should be so calls for further study.

In the semi-arid Northwest Cape Province, the iodine content of drinking water is high, fluorine is high and it has been reported by Malherbe and Ockerse[5] that the calcium, magnesium and sodium content is also high. The authors of the report ascribe endemic goitre in this situation to harmful amounts of fluorine in the water. In 1941 Wilson[9] came to a similar conclusion about goitre in the Punjab and parts of the United Kingdom, but made no mention of other minerals. In the South African report the

possible role of excess calcium as a goitrogen is mentioned, but more importance is attached to fluorine excess. One feels that the fluorine theory should be tested again by more extensive animal experiments, with due reference to analysis of variance techniques.

The absence of cretinism in South Africa even in the worst endemic goitre areas is commented upon in the report.

It has been known for years that a cabbage diet may induce goitre in rabbits.[2, 3] Various other members of the *Brassica* family may have a similar effect, also in man.[4] In 1936 Barker[1] found that certain of his hypertensive patients, whom he was treating with potassium cyanate, developed enlarged thyroids and myxœdema. Cabbage contains thiocyanate, so there is a definite common factor between the cabbage-eating rabbit and the hypertensive patient. As a matter of interest, it may be mentioned that Silink and Marsikova,[7] working in Czechoslovakia, found a relatively high blood thiocyanate level in the blood of people living in an endemic goitre area. It is suggested in the present report that the eating of hydrocyanate containing cassava may aggravate the endemic goitre situation in the Eastern Caprivi.

The authors of the report recommend the use of iodized salt at the level of one part of iodine per 100,000 parts of salt in the iodine deficient areas, except where endemic goitre is attributed to excess of fluorine. In these areas the removal of fluorine from water supplies appears to be the solution, an almost impossible task at present. For iodization the use of potassium iodate (KIO_3), rather than potassium or sodium iodide, is proposed, as the former is more stable than iodides.

What emerges from this report is that considerably more detailed studies are now required in South Africa, and elsewhere, of goitrous patients in relation to their total environment. It is not enough to test soils, water and food only for iodine and fluorine; they must be examined for other elements besides. It is not enough to examine only the thyroid glands of patients; there should be more complete clinical and biochemical studies. But this is perhaps a counsel of perfection not attainable in an imperfect world.

The authors of the report are to be congratulated on their work and we hope to hear more about this interesting topic from South Africa, a country abounding in so many different physical environments and with so many racial groups living in conditions ranging from primitive poverty to urban luxury.

W. HARDING LERICHE

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THE NATURAL HISTORY OF PERFORATED ULCER

Eleven years ago, some Scottish surgeons published a 20-year survey of acute perforated peptic ulcer in the West of Scotland. One of this group (Jamieson, R. A., *Brit. M. J.*, 2: 222, 1955) has now extended the survey to cover the period 1944 - 53. The sample, which includes a large Western Scottish area, amounts to 6,343 proven perforations, usually operated on by simple closure of the perforation.

This catastrophe has become increasingly common over the last 30 years, the annual incidence per 100,000 population rising linearly from 11 in 1924 to 33 in 1953, with an abnormal hump during the war years. Males are greatly preponderant, but not so much in recent years; the great majority of patients who perforate are middle-aged, and the mean age at perforation is slowly rising. It may be, of course, that ulcer patients are surviving longer and having a chance of perforation at a later age. Each year, about 1 - 2% of adult males with ulcer have a perforation, and the risk of a second perforation appears to be of the same order.

Jamieson notes that there is a fall in incidence of perforation in August, September and October (possibly associated with the fact that July is a holiday month in the West of Scotland) and a peak in December, when the artisan class tends to overwork. As further support for the hypothesis that fatigue is associated with perforation, the author shows that the incidence rises steadily from Tuesday to Friday, with a marked low level on Sunday; moreover, perforation is relatively uncommon at night, rising in incidence to a minor peak before lunch and a major peak at the end of the afternoon.

Pyloro-duodenal perforations far exceeded all other types, which is fortunate in view of the high mortality of gastric perforation. The general fatality rate has continued its fall from 25% in 1924 to 8%, in spite of the increasing number of elderly persons involved with a much higher mortality. The usual sharp rise in fatality with delay in treatment was seen, and also a tendency for fatality rates to increase in winter, possibly due to pulmonary complications.

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PUBLIC RELATIONS FORUM

Conducted by L. W. HOLMES,
Assistant Secretary, C.M.A.

II. GATEWAY TO GOODWILL OR GRIPES

THE REMAINING ARTICLES in this series will stress the *How* of medical public relations. The next several discussions will focus on the relationship between individual doctor and individual patient —the foundation of good public relations. These will follow the patient through his many dealings with the doctor and will mark the attitude-shaping milestones along the way. Subsequent articles will trace the avenues of public relations service open to organized medicine.

ENTER THE PATIENT

The first thing the patient sees when he visits the doctor is the reception room. This, then, is the first impression-forming factor to which the doctor should turn his critical attention. He might well take a leaf from the homemaker's work book. The housewife takes pride in her home. She wants it to be neat, clean, genial and comfortable. She wants it to be home to her family and homelike to her visitors.

The doctor should want his patient to feel at home when he visits the office. His reception room should be designed for patient welfare and comfort, and at the same time reflect the doctor's personality, to give the impression that he really belongs in that setting.

No patient, wrapped in his own distress, his pain, his fears, wants to enter dreary and depressing surroundings. He needs to be reassured, to be cheered, to be treated as something special. The first step in that direction is a comfortable, tidy, attractive, bright reception room.

The reception room doesn't need the professional decorator's touch. It doesn't need expensive furniture, floor coverings or draperies. All it needs is colour harmony, bright but not garish.

Furniture should be comfortable and adequate for the number of waiting patients at peak periods. It should be arranged carefully; the finest collection of furniture will serve little end if it is arranged without purpose. Placing chairs around the room so that patients face each other is unwise. Misery is infectious, and the sick person has enough of his own without having to share his neighbour's. Furniture should be set out to provide convenience in reading, conversation and writing. The provision for writing is an "extra" which patients will appreciate.

Lighting, ventilation and heating are matters which require careful attention. Lamps should be placed for maximum reading ease. If smoking is permitted, ventilation should be adequate to protect those who might be offended by tobacco smoke. Reception room temperature should be warm enough to encourage patients to remove their outer coats but not warm to the point of discomfort. And once the coats have been removed there should be some place to put them. Storage space for hats, coats, umbrellas and rubbers will be appreciated.

A word about reading material. The modern doctor subscribes to a number of magazines and if he is patient-comfort conscious he will see that only the recent, less dog-eared copies are placed conveniently around the room. If health literature is put out, it should be chosen with care; it should be constructive, not destructive.

The foregoing describes the bare minimum toward which every doctor should strive when examining his reception room PR. But the progressive doctor is always on the watch for the "extras" which will help win patient-friends. The doctor who insists that his reception room is, in reality, a living room, will hang pictures and provide fresh flowers and plants. Some doctors have installed television sets; some pipe in soft music. A few doctors have established children's corners to relieve harassed mothers. One doctor, apparently a firm believer in health education, has installed tape recorders in special cubicles. These machines play health messages while synchronized projectors illustrate the lectures. Another has put in a motion-picture projector.

In busy downtown areas, where parking facilities are limited, some physicians have arranged for parking space close to their offices. This helps eliminate the patient's complaint that he received a parking ticket while waiting to see the doctor.

Whether he meets the bare essentials for patient comfort, or searches for the extra touch, every effort to provide a pleasant, cheerful waiting-room is a sign to patients that the doctor has their welfare at heart. The payoff comes in pleased patients, a major step in the direction of excellent public relations.

Doctor: Have you looked at your reception room lately—looked at it, that is, with the critical eye of the patient? Is it all it should be? Is it a credit to your practice? If not, you've been passing up an opportunity to serve your patients better, and at the same time, the profession as a whole.

PR TIPS

Opening a stack of bills every month can be pretty unpleasant, and this applies to doctor bills, too. To make their bills a little less distasteful, many institutions (telephone companies are good examples) include with their billing a short message, an item of news, a word of advice, a friendly greeting.

Why not the same technique for cementing good patient-doctor relationships? There are a great many practical messages that the doctor can send to patients. These may take the form of a personal letter, a circular letter, a printed folder.

On July 25, 1952, Dr. Bernard P. Harpole, of Portland, Oregon, started a letter to his patients which he sent out with his monthly bill. In it he gave health hints, discussed medical economics, extended season's greetings, suggested what to do in emergencies, and how patients might help him improve his service, etc. A similar approach may provide Canadian doctors with a PR medium. Interested readers may obtain copies of some of Dr. Harpole's letters from Public Relations Forum, Canadian Medical Association, 244 St. George Street, Toronto 5, Ontario.

Men and Books

JOHN HILTON: "REST AND PAIN"

ALEXANDER GIBSON, F.R.C.S.(Eng.),
Winnipeg, Man.

DURING THE YEARS 1860, 1861 and 1862, a series of 18 lectures was delivered at the Royal College of Surgeons of England by John Hilton, "Surgeon Extraordinary to Her Majesty the Queen, Consulting Surgeon to Guy's Hospital, late President of the Royal College of Surgeons of England, Etc., Etc., Etc." The title page of the volume in which these lectures were published reads as follows: "On Rest and Pain, a Course of Lectures on the Influence of Mechanical and Physiological Rest in the Treatment of Accidents and Surgical Diseases and the Diagnostic Value of Pain."

Although it is nearly a century since this book appeared, there are several reasons why the student and the practitioner of today should make its acquaintance. It is true that in those days physiology was elementary, biochemistry was primitive, bacteriology was not yet born and radiology was undreamed of. But anatomy was studied and pondered over with a thoroughness that is no longer deemed necessary, and clinical observation was the key to medical progress. Life was more leisurely in those days; there were fewer extraneous distractions, there were not so many competing avenues of research, there were no short cuts to diagnosis such as electrocardiography or myelography. The doctor depended for enlightenment mainly on his five senses and his accumulated experience. Today, in the larger centres at least, although we continue to pay lip-service to clinical observation, the practice of medicine has become more and more mechanized. The gain in objective accuracy is very considerable (and very expensive), and yet we have lost something too. When we peruse the writings of the great clinicians of the past, we cannot but admire the amount they accomplished by the use of their eyes and ears and by thinking over what they observed. Much escapes our notice when we are jet-propelled.

The more popular "Histories of Medicine" give little information about John Hilton. For much that follows I am indebted to the graphic pen of Sir Arthur Keith. Hilton was born in 1807 in North Essex, of pure Saxon stock. At the age of 17 he went to London and attached himself to Guy's Hospital in the Borough. In 1828 he became a demonstrator of anatomy, and in the dissecting room he remained for the long period of 17 years. In 1845, when he had reached the age of 38, he was appointed to the posts of assistant surgeon and lecturer on anatomy. These were the days before Darwin's "Origin of Species" had introduced the idea of evolution to the scientific world. The dominant influence in biological thinking was that of William Paley, an Anglican divine. In 1794 Paley published a celebrated book, "View of the Evidences of Christianity", and in 1802 there appeared "Natural Theology or Evidences of the Existence and Attributes of the Deity Collected from the Appearances of Nature." These volumes are still obtainable in the second-hand bookstores in Great Britain. Paley laid stress on the manifestations of design, especially in human anatomy. Each part was created for a specific purpose. Usually the word "teleological" is employed to describe this viewpoint, generally with a little raising of the eyebrows. It is, however, nowadays quite praiseworthy to point out the correlation between form and function, provided one does not insist that the relationship is one of cause and effect. Hilton's view of anatomy was frankly teleological so that he could describe the subclavian artery and its branches as the "artery of respiration", and the cœliac as the "artery of digestion." The nerves of the body were thought of in a similar manner. In Lecture VII he says: "In order to bring in a comprehensive and definite form before you this fact, which is so important on anatomical, physiological, and pathological grounds, I will state it thus:

> The same trunks of nerves whose branches supply the groups of muscles moving a joint furnish also a distribution of nerves to the skin over the insertions of the same muscles; and—what at this moment more especially merits our attention—the interior of the joint receives its nerves from the same source.

This implies an accurate and consentaneous physiological harmony in these various co-operating structures."

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"The object of such a distribution of nerves to the muscular and articular structures of a joint in accurate association is to secure mechanical and physiological consent between the external muscular or moving force, and the vital endurance of the parts moved, namely, of the joints, thus securing in health the true balance of force and friction until deterioration occurs." The importance of this principle is illustrated in the actions of standing, walking, jumping, and prehension.

In these pre-Listerian days, a large part of surgical practice consisted in opening abscesses. Lecture VI is devoted to this subject. A quotation from the summary at the head of the chapter reads as follows:

"Abscesses opened to secure co-aptation to their internal surfaces and to permit their union by giving them rest—Principle exemplified in sub-mammary, knee-joint, axillary, orbital, cervical, post-pharyngeal, iliac, subgluteal, subfascial, and sub-muscular abscesses, together with the best method of opening an abscess . . ." The author lays stress on the necessity of opening an abscess at its lowest point if adequate drainage is to be secured. It may be interesting to quote his own description of the method of evacuating a collection of pus which, to this day, is spoken of as "Hilton's method":

> The plan I have been in the habit of adopting and recommending is this;—in the case for example of opening a deep abscess in the axilla—cut with a lancet through the skin and cellular tissue and fascia of the axilla about half or three-quarters of an inch behind the axillary edge of the great pectoral muscle. At that part we can meet with no large blood-vessel. . . . Then push a grooved probe or grooved director upwards into the swelling in the axilla; and if you will watch the groove in the probe or director as it is being passed up through the comparatively healthy tissues into the axilla, a little stream of opaque serum or pus will show itself. Take a blunt (not a sharp) instrument, such as a pair of dressing forceps, and run the closed blades along the groove in the probe or director into the swelling. Now, opening the handles, you at the same time open the blades situated within the abscess, and so tear open the abscess. Lastly, by keeping the blades of the forceps open during the withdrawal of the instrument, you leave a lacerated track or canal communicating with the collection of pus, which will not readily unite and will permit the easy exit of the matter. In this way you may open an abscess deep in the axilla or in other important parts of the body, without fear of inflicting any injury upon the patient.

There follow case-histories to illustrate the use of the method in different situations. All are related in a brisk and lively style, with more than one touch of sly humour.

Hilton was essentially a conservative surgeon. He was a staunch believer in the *Vis medicatrix naturæ*. "I feel convinced that, under the most favourable circumstances, all that any of us can accomplish is to give rest to the parts, and enable Nature, through her own efforts, steadily to play her part, while we, as Nature's willing servants, act in the hope that . . . . we may facilitate her efforts to repair the injury she may have sustained. In fact, nearly all our best-considered operations are done for the purpose of making it possible to keep the structures at rest, or freeing Nature from the disturbing cause which was exhausting her powers, or making her repeated attempts at repair unavailing." A cheery little anecdote related in Lecture III illustrates his fundamental viewpoint.

> A few weeks ago a surgeon from the country came to my house with a patient. He said, "I want to consult you about a young lady who has a diseased toe." With her was a relative, an elderly gentleman, a very kind-hearted man, who thinks himself a good surgeon, and goes about doctoring people, sometimes doing harm, and sometimes, perhaps, a great deal of good. He is very fond of animals and has a number of pets. After I had examined, with the surgeon, the lady's toe, the elderly gentleman said, "Well, Mr. Hilton, what are you going to do to cure this young friend of mine?"" I said, "I think we shall put a splint on the foot and keep the toe very quiet, attend to her general health, and Nature, in all probability, will do the rest." I then said to him, "What led you to adopt the occupation of a philanthropic surgeon in addition to your other occupations?" "Well, Mr. Hilton," he replied, "I will tell you. You know I am very fond of animals. Some years ago I caught a live mouse in a trap. I took it in my hand, and I said to myself, 'Poor thing, you must have suffered a good deal. You have had a severe laceration of your cheek; one of your eyes has been torn out; your skull has been broken and, instead of having bone covering your brain, you have now only a thick, dense membrane defending it.' Then I thought to myself, 'This mouse must have had difficulties in the treatment of its injuries; and,'"—interrupting the relation of his story he said, "I hope you won't be offended at what I am going to say?" "No", said I, "not in the least." "Well," he continued, "I said to myself, 'Surely this mouse, although it is cured, never had a physician or a surgeon!' I quite agree with you, Mr. Hilton, that Nature is a very valuable surgeon."
>
> It would be well, I think, if the surgeon would fix upon his memory, as the first professional thought which should accompany him in the course of his daily occupation, this physiological truth—that Nature has a constant tendency to repair the injuries to which she may have been subjected, whether these injuries be the result of fatigue or exhaustion, of inflammation or accident.

That the term "rest" was to be interpreted in the widest possible way is indicated in Lecture XI. "A physician residing not very far from me had under his care a patient who had received a blow on his chest by a fall upon the part; and as he was after several days, still suffering a good deal of pain in breathing, the physician asked me to see him in reference to the possibility of fractured ribs. I could find no fracture; but I observed that the patient had a most worrying wife. She was incessantly talking to him day and night, and there were continued contentions between them upon domestic affairs. I suggested to the physician that the sole cause of the pain was in all probability produced by the patient constantly moving the injured or bruised soft parts by using his chest and lungs in speaking. All I recommended was that he should hold his tongue and have his chest bandaged. I requested that his wife would not say

a word to him but would provide him with a slate and pencil so that he might write down all his desires. From that time he got quickly well *by local rest.*" The last three words are italicized in the published account; I rather think that Hilton was smiling to himself as he wrote them down.

Somehow or other the name of Hilton has been identified with the doctrine of rest, without taking into equal account his teachings on pain. In Lecture VII, he refers to pain as "Nature's warning prompter"; on every occasion he lays stress on the necessity for finding out exactly what the lesion is that is responsible for the pain. The following paragraphs are not so very old-fashioned.

> When a patient complaining of pain applies to a surgeon, the surgeon ought to seek for the real cause. He ought not to be satisfied as is too frequently the case with saying, "Oh, it is rheumatism" (the favourite phantom). "You have caught a cold." "You have been standing in a draught of air" . . . "it is the easterly wind which has been lasting so long; wait till the wind changes." "It is gout." The patient says, "It cannot be; I live so carefully." "But," says the surgeon, "you have inherited it from your father or your great-grandmother; or you must have had a blow on the part some time ago, which you do not recollect—that is all."
>
> Now, external pain or pain upon the surface of the body if properly appreciated, may be considered as an external sign of some distant derangement. If the pain persists,—if it does not depend on any transient cause—it becomes necessary to seek the precise position of the pain; and as soon as we recognize the precise position of the pain, we are enabled, by a knowledge of the distribution of the nerve or nerves of the part, to arrive at once at the only rational suggestion as to what nerve is the exponent of the symptom. By following centripetally the course of that nerve, and bearing in mind its relation to surrounding structures, we shall—in all probability—indeed most likely be able to reach the original, the producing cause of pain, and, consequently, to adopt the correct diagnosis.

The use of the affected nerve as an indicator of the region involved is illustrated by several case-histories, driving home the point that it is necessary to determine exactly and minutely the particular nerve involved. Hilton was ever alert to increase the preciseness of his anatomical knowledge.

> A short time since, a man, who is now undergoing the punishment of penal servitude, attempted to cut his wife's throat. In drawing the razor across her neck, he divided the auricular branch of the second cervical nerve, and gave me the opportunity of ascertaining the distribution of that nerve. My dresser, as well as myself, pricked with a needle over the whole of the auricular surface, and ascertained minutely the precise position of the loss of sensation consequent upon the division of the cervical nerve; while the skin which retained its sensation indicated with equal precision the distribution of the fifth cerebral nerve upon the external ear.

Lecture V is devoted entirely to the subject of "pain as a local symptom in relation to diseases of the spine". "I will, for the sake of brevity, endeavour to reduce my views to the form of a proposition. I would state then: that superficial pains on both sides of the body, which are symmetrical, imply an origin or cause, the seat of which is central or bilateral; and that unilateral pain implies a seat of origin which is one-sided, and as a rule, exists on the same side of the body as the pain." Illustrative cases are cited, e.g., "Case of diseased spine with symmetrical abdominal pains". "Disease of the spine with pains in the back of the head". "Disease of the spine with pain at the back of the head and over the left shoulder and in the left arm." "Disease of the spine with loss of power and sensation in the limbs". "Case of diseased spine; sudden death of the patient (from pressure by the odontoid process of the axis upon the medulla)." "Case of diseased spine, with post-pharyngeal abscess, from which were expelled portions of the atlas and axis". So much progress has been made in the diagnosis and treatment of tuberculosis that examples of the clinical conditions described by Hilton are unlikely to be met with nowadays except in remote parts of the country, for instance, among Indians and Eskimos.

Lectures XIV and XV are devoted to disease of the hip-joint; most of the cases described are undoubtedly examples of tuberculosis, yet the opening sentences make it very clear that the author was little inclined to accept all lesions of the hip-joint as "scrofulous." "If the surgeon affix such an idea to every case, or to the majority of cases of hip-joint disease coming under his notice, he will feel little disposed to adopt anything like a persevering plan in his practice, and without such a plan he will surely fail in the proper treatment of the disease." One figure portrays a splint devised by Hilton for the treatment of a little girl aged seven. "When admitted into Guy's in June 1860, she had all the indications, local and general, of severe hip-joint disease with deep suppuration in the anterior, upper and outer part of the thigh. The limb was flexed and adducted; the patient was suffering great pain in the hip, had little sleep at night, and little or no appetite. Chloroform was administered, and the contracted muscles of the joint yielded steadily and nicely to carefully applied extension. The flexed limb was made straight, and a long common iron splint applied along its outer side extending from near the axilla to the foot with a transverse bar to prevent rotation. Immediately after the application of the splint, all the disturbing symptoms began to disappear and her appetite returned. In February 1861, there was not a single symptom." Ankylosis had occurred.

Lecture XVIII is the last of the series, and in it the last case he describes is interesting as a sidelight on Hilton's enthusiasm for getting to the root of things, and also as a contrast to present-day funeral arrangements.

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A young gentleman, eighteen years of age, tolerably healthy, and living at Islington, was always active and fleet of foot. On Friday, January 10, 1862, he ran two miles right off to a friend's house, and, after resting for a short time, he ran two miles back home but suffered no known inconvenience from that exertion. On the Sunday evening following, returning from chapel, wearing a very narrow, high-heeled boot, and walking at the edge of the pavement, his left foot turned inwards with a sudden jerk or twisting sensation, and he exclaimed to his sister, who was with him, "Oh, I have twisted my foot; I never had such a dreadful wrench before." He walked home, and, excepting at the time of the strain, he felt but little of the injury during that evening or night. He walked to his business—10 minutes walk from his home—on Monday morning, when the pain in his leg near the ankle became so great that he returned home; he limped, and could scarcely bear any weight on the foot."

A week later the patient was seen by Hilton who "believed the soft parts between the shaft and the lower epiphysis of the tibia were injured . . . he had a small collection of pus deep in the leg at the inner and back part of the tibia. I did not again see this patient alive. Five or six days afterwards he died with what was thought to be typhoid fever."

By chance on the day of his funeral, I heard of his death. I immediately wrote to his relations, and obtained permission to make an examination of the leg. I went to the house early on the following morning; the hearse was at the door, so that I had only time to unscrew the coffin and examine the leg. The periosteum was separated from the inner, anterior, and the posterior parts of the lower portion of the tibia by a considerable collection of pus, which was confined in its position by the periosteum. I took away the bit of the tibia, and it fairly remunerated me for my trouble.

It may be worth while to quote the concluding paragraphs of the book in which he summarizes his aims and his frame of mind:

I trust I have neither dogmatized nor spoken presumptuously . . . I have herein endeavoured to show that "Rest" is a most important therapeutic agent in the cure of accidents and surgical diseases. To illustrate the varied applications of this principle, I first surveyed, as fully as my limits permitted, the marvellous contrivances which Nature has employed for securing rest to the different organs of the body when in health. I then depicted the instinctive promptings of Nature to secure 'Rest' on the occurrence of accident or disease. Lastly, I attempted to shadow forth the different appliances for the attainment of rest with which that surgeon only will become familiar who has an accurate knowledge of the anatomy and physiology of the different parts which he may be called upon to treat.

I have also endeavoured to impress upon you the fact that every pain has its distinct and pregnant signification, if we will but carefully search for it. To the extent of my present opportunity I have striven, by the agency of a more precise nervous anatomy, to unravel and render patent the meaning of pains which have been so often described as *anomalous or obscure*. From the pain which follows the intrusion of a particle of dust on the conjunctiva and the closure of the eyelid for the security of rest, up to the most formidable diseases we have to treat, pain the monitor, and rest the cure, are starting points for contemplation which should ever be present to the mind of the surgeon in reference to his treatment.

I trust that in the foregoing lines there has been presented a trustworthy picture of this remarkable book, and to some extent of the man who wrote it. Some books live because of the thesis they elaborate; others because the personality of the author shines through them. *Rest and Pain* lives for both reasons. It will continue to live for an even simpler reason; it is readable.

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GENERAL PRACTICE

THE FAMILY PHYSICIAN: A VANISHING CANADIAN?

W. HARDING LERICHE, B.Sc., M.D., M.P.H.,
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THE TREND towards increasing specialization in medicine during the last 30 years has had great advantages, especially in a technical sense. Incredible surgical procedures are undertaken successfully and rare conditions in the field of endocrinology are ceaselessly studied and elucidated. The common infectious diseases can be prevented or relatively easily cured. Even poliomyelitis is yielding to the Salk vaccine and developments in virology.

But, as in other fields, technology has outstripped social organization. People still live in groups. They still have families. And even in cities, people like to know their doctor, traditionally a man or woman who has known their particular family over a long period of time. He has always been, and will again be, a rock standing safe in the shifting sands of urban social change.

This is no plea for a return to the horse-and-buggy days. The horse and buggy has now become a flashing automobile. The old physician's single ledger has now become an imposing filing cabinet. The small office has become a big office. The common cold has become coryza. The hungry baby of the overanxious mother has become the hypertonic infant. But people have remained the same. And now, even more than in the past, they need one interested physician who will guide them through the maze of medical technology, and who will provide genuine interest and friendship which means so much more than techniques.

To be a good general practitioner is a great achievement. It has been said with some truth that, lacking the versatility essential in a good general practitioner, many physicians have to become specialists, with a more restricted field. And in certain instances, more technology and gadgets may at times mask defects of temperament and personality which would be more apparent in general practice.

There are many good general practitioners in Canada today, but their numbers seem to be relatively decreasing. For instance, in June 1951 there were, according to statistics of the Research Bureau of the Department of National Health and Welfare, 604 general practitioners in Toronto. Three years later there were 606 general physicians in Toronto. During the same period specialist physicians in the metropolis increased from 578 to 660. These figures for physicians in private practice adequately parallel increases in population, but to the public there are now fewer physicians available who will do house calls and who will attend to emergencies, because most specialists work only during office hours.

There is no point in blaming the medical profession for this situation. It is the result of the interaction of a number of different forces, some from the public, and others from the profession. In general, many specialists have regular hours and good incomes. Some have very high incomes. Physicians would like to enjoy these advantages and therefore become specialists. People go to specialists because they want the best in medical care. But the specialist usually gives only partial care: to an eye, a nose, a skin, an appendix, not to the whole patient. It is a moot point whether three or four or five specialists, not one of them knowing a family, can provide better care than one competent family physician who will decide when the patient needs specialist assistance. The specialist care will cost more than one general practitioner's. To some people in a materialistic age, this might provide cause for satisfaction as an indication of their personal financial status, but their health will not necessarily be improved thereby. If good health were solely dependent upon the activities of good technicians, like good mechanics who can keep an automobile in perfect order, then the more specialists per human body the better. But things do not work that way. Our old-fashioned bodies are not merely sentient machines. A heart is not merely a blood-pump, the muscular contractions of which may be partially interpreted in terms of galvanic currents of an electrocardiograph. That heart will react to disease, but as surely it will react to emotions—to feelings, to love and hate and fear. Physicians, like mothers and fathers, aunts and uncles, teenagers and infants, parsons, priests and politicians, are part of our old-fashioned social organization, itself full of love and hates and fears. The family physician is part of this social scene.

It is clear that we need specialists. But more than these we need one type of doctor, perhaps another kind of specialist in this age of specialists, who will handle most of the ills of one family, because the ordinary illnesses can all be handled by one physician. Some of the most difficult family situations can only be treated and alleviated by a general practitioner.

The people need good general practitioners. The medical schools should produce such practitioners. If the universities are not doing so at present, their orientation should be changed so that they can do this essential job. The Canadian College of General Practice is at present actively concerned with improving both the training and the performance of the general practitioner so that in the future there will be no doubt that the family physician, with the latest knowledge and technical equipment, will increasingly take over family medical care again, referring special cases to specialists.

If more physicians with specialist training in various fields take up general practice, this will be all to the good, as it will greatly improve standards of technical performance. There are already signs of such a development in Canada.

393 Ruth Avenue.

PRINCE EDWARD ISLAND CHAPTER

THE ANNUAL MEETING of the Prince Edward Island Chapter was held on August 3, 1955, at the Charlottetown Hotel with Dr. L. G. Dewar, chairman, presiding.

The afternoon business session received reports from Dr. Henry Moyse, chairman of the education committee, and Dr. Dewar on the annual meeting of the College in June in Toronto.

A dinner meeting was held at 6 p.m., after which a clinical session was held with the following speakers taking part: Dr. Henry Moyse on Cortisone Therapy; Dr. Owen Curtis, Use of Gamma Globulin in Polio; Dr. Wm. Moreside, Glaucoma; Dr. Joseph A. MacMillan, Trans-Canada Medical Plans; Mr. Jelks, The Use and Abuse of Antibiotics; Dr. Daniel Tonning, Acute Methyl Alcohol Poisoning.

The following officers were elected for the coming year: Chairman: Dr. T. L. Farmer, Charlottetown. Chairman-Elect: Dr. R. A. Reid, Wellington. Secretary: Dr. W. E. Callaghan, Summerside. Treasurer: Dr. H. P. Stewart, Charlottetown. Representative to Board of Representatives: Dr. L. G. Dewar, O'Leary.

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MEDICO-LEGAL

THE CONDUCT OF A TYPICAL MALPRACTICE ACTION*

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I WOULD LIKE to try to trace for you the various steps in a typical malpractice action and to offer some suggestions which may be of assistance to you in the event that you find yourselves in the unfortunate position of being involved in one. Before doing so, however, there seemed to me to be some merit in outlining briefly the duty which the Courts impose on you as practising doctors. A clear and concise statement of the law and one on which I cannot improve is found in an article by Mr. Edson L. Haines, Q.C., entitled "Courts and Doctors" and printed in the May 1952 issue of the *Canadian Bar Review*. Mr. Haines, in writing of the medical man's standard of care, says:

> The essential obligations the law imposes upon the physician or surgeon in general practice who takes charge of a case are simply stated. They are:
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> 1. He must possess and use that *reasonable* degree of learning and skill *ordinarily* possessed by physicians and surgeons in the locality where he practises. The key words in this statement are "reasonable" and "ordinarily". The degree of learning and knowledge is the degree that might reasonably be expected from the average doctor in the district. Few men have rare and extra endowments, but a doctor is not judged by the standards of the paragon. He need only display the skill and learning of the average physician. Furthermore, a country practitioner, for example, may not have the same opportunities to confer with fellow practitioners as the practitioner in larger centres and one must take into consideration, therefore, the locality where the doctor practises.
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> 2. The doctor must use his best judgment. Judgment is the faculty of deciding wisely. The law will not be satisfied with anything less than the use of "his best judgment" in exercising his skill and applying his knowledge. He must make the best and wisest decision within his power. It does not of course require him to have or use the best judgment some other man might use. It is not the best *possible* judgment, but *his* best judgment, he must bring to bear.
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> 3. He must keep abreast of the times and follow the approved methods in general use. He must know what is going on in medicine, what new discoveries have been made, what old opinions or conclusions have been discarded. He should take advantage of medical and scientific journals and, if possible, attend conferences where he may exchange information with other physicians. Doctors are singularly fortunate in the wealth of scientific literature which is available to them and they fail to take advantage of it at their peril.

THE SPECIALIST'S STANDARD OF CARE

I have already pointed out that the standard of care of the average general practitioner is referable to the locality in which he practises. Conceivably, it might vary in different districts. There is no such local standard for the specialist. He holds himself out as possessing special skills and knowledge, and it is his duty to have and apply the degree of skill possessed by the average specialist in his field. The same degree is expected of him whether he is practising in a large city or a small centre.

The Honourable Mr. Justice Smily in the case of *Gent v. Wilson* expresses it in this way:

> I have intimated in the course of the argument that one could not help feeling sorry for not only the infant plaintiff, but for the parents for the very great anxiety which the parents must have had and for the suffering which the child must have had as a result of the course of this ailment; but it is recognized, and I suppose counsel for the plaintiffs would not quarrel with the statement, that because a course of illness or medical treatment has had untoward results that does not imply that there has been any, not only negligence but any failure of any kind on the part of the medical practitioner in respect of his treatment and conduct as a professional man. Doubts may very well be raised in the mind of a person who has had an unfortunate medical experience and result from vaccination and otherwise and also in the mind of parents with a child, but that of course does not mean that there has been fault or failure on the part of the medical practitioner. The professional person does not insure cures or even satisfactory results or even, you might say, the expected results. All he can do is do the best he can to carry out the professional service in a proper and recognized professional manner with the care which is required in such matters. There is always the hope that that will bring about a proper result, a cure, or a satisfactory result, but sometimes that does not happen; but, as I say, the fact that it does not happen does not mean that the doctor has not used the proper treatment or used proper care.

As you will see from the foregoing, the duty imposed on a doctor is a heavy one, but it is also clear that the law neither expects nor requires a doctor to perform miracles. At the same time, in any action for malpractice it is incumbent upon the doctor to demonstrate to the satisfaction of the Court that the duty imposed upon him by the law as outlined above has been met. How one goes about proving this will be discussed a little later. If you have been obviously careless in the treatment which you have prescribed or if you have ignored what is generally accepted to be good, sound medical practice in all the circumstances of a particular case, you will not have the support of your fellow practitioners when that support is most urgently needed and consequently your task of proving that you were not negligent will be immeasurably greater.

Let us now trace an action from the moment of its inception to, we hope, its successful conclusion. Except in those cases where the very circumstances themselves make it perfectly apparent that future legal difficulties will be encountered, the first intimation of trouble may not reach you for some months after the matter complained of has been completed. In most provinces of Canada an action for malpractice must be begun within one year from the date

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*An address given at the annual meeting of the Canadian Medical Protective Association, held in Toronto on June 22, 1955.

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on which the services of the physician in the matter in question terminated, but it is not unusual to find a plaintiff neglecting to make any demand until the period of the year has almost expired. This delay, and there will be others before the action is concluded, leads me to the first suggestion which I would like to make to you. Altogether apart from medical considerations, it is extremely desirable that full and complete notes be maintained by every doctor on every patient. I realize that this must be one of the greatest chores facing any practising physician, but nevertheless it is one which in your own interests you cannot afford to ignore. Memories and recollections become dulled with the passage of time, and unless your records made at the time of treatment or shortly afterwards are detailed and accurate, you may very easily find yourself in the position of being unable to recall with certainty the exact treatment prescribed or the symptoms observed at the very moment when it is most important that you should be in a position to do so.

Now that your patient or ex-patient has communicated with you, either directly or through his solicitor, and either orally or in writing, complaining of the treatment which you prescribed and alleging that for one reason or another you were negligent in the circumstances in question, you should write immediately to the Secretary-Treasurer of your Protective Association informing him of the threat and giving him a detailed medical history of the case. Do not answer the enquiry until you have received a reply to your letter. Do not be afraid to include in your letter the most minute details of your treatment even though such details may seem to you to be so obvious that they can be taken for granted. Bear in mind that the men considering your letter are all outstanding physicians but that they were not with you when the treatment was given and their only knowledge of what you did and why you did it is what you state in your letter. The fact that you omit a part of the treatment prescribed may lead them to believe that you did not in fact give such treatment, and in the particular circumstances such an omission might very easily be considered as negligence. For instance, if anti-tetanus serum is indicated in a particular case, do not assume that other practitioners will conclude that you did in fact give the necessary injection. They may, and very probably will, conclude that your failure to mention it means that you did not give it and might quite rightly determine that failure to do so was negligence on your part. The whole conduct of the action will have been prejudiced because of the omission of a detail, thereby resulting in the drawing of an erroneous conclusion. At the best, time is wasted while enquiries are made; at the worst, decisions are made based on a false conception of the facts.

Having received the advice which you sought from Ottawa, it is unnecessary to suggest that you should follow such advice as closely as possible. It may transpire that nothing will be heard of the proposed action, but the likelihood is against this and in any event if we are going to follow an action through, we must assume that it does not stop with the original demand.

In order to begin proceedings in Court the plaintiff through his solicitor prepares what is known as a Writ of Summons. A Writ of Summons is in effect an Order of the Court—normally in a malpractice action, the Supreme Court of the Province, commanding the person named in the Writ as the defendant to appear to answer the charges made against him by the person making the claim, called, in Court proceedings, the plaintiff. While the Writ is an Order of the Court, it is prepared and issued by the plaintiff's solicitor in his own office and not by the Court officials. Therefore, the claim as contained in the Writ is simply a short statement by the plaintiff's solicitor indicating the nature of the action, and, generally speaking, gives the defendant little or no information as to the underlying facts behind the cause of action. In the absence of special authorization by the Court, the Writ must be served on the defendant personally, although the defendant's solicitor, if so authorized by the defendant and provided he is prepared to undertake to protect the defendant's interests, may accept service on his behalf. In the event someone should attempt to serve a Writ on you, it is both futile and unwise to try to avoid such service. In the first place the person trying to serve you will undoubtedly persevere until he has been able to effect his purpose and, in the second place, if you are successful for any period of time in avoiding service there are procedures open to the plaintiff whereby substitutional service may be made. At a later stage in the action a good lawyer could conceivably make capital out of the defendant's efforts to avoid service in the first instance. For these reasons you should accept service of the Writ without comment and once again immediately contact the Secretary-Treasurer of your Association. Counsel will be retained on your behalf and you will be instructed to send the Writ to him and to co-operate with him in every way in the preparation of your defence and the conduct of the action. From this point on you are really in the hands of the lawyers.

If you read the Writ, you will see that it contains a statement to the effect that, within a prescribed number of days from the date of service of the Writ upon you, you must cause an Appearance to be entered in your behalf and that in default of such Appearance the plaintiff may sign default judgment against you. It be-

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comes obvious that time is an important factor at this particular stage and immediate action on the Writ is necessary in order to protect your interests. The Appearance referred to in the Writ is not a personal appearance; it is merely a very short document prepared by the defendant's solicitor and filed with the Court, indicating that you have entered an Appearance in the action and therefore by implication confirming that you wish to defend the action.

When the plaintiff's solicitor sees that an Appearance has been entered, his next task is to prepare and serve on the defendant and file in the Court a document known as a Statement of Claim. In the Statement of Claim the parties to the action are identified, the facts giving rise to the cause of action in so far as they are within the plaintiff's knowledge are set out, the negligence alleged is indicated, and the damages claimed as a result of such negligence are stated. The amount of damages claimed in the Statement of Claim will probably bear no relationship whatever to the damages which the plaintiff may hope to recover and almost invariably will be considerably greater than the latter figure. The reason for this is that, generally speaking, a plaintiff cannot recover higher damages than he has claimed in his Statement of Claim and consequently the solicitor in preparing the Statement of Claim always plays safe by asking for damages greatly in excess of what he actually hopes to recover for his client.

It is now the responsibility of the defendant to answer the allegations made by the plaintiff in the Statement of Claim and this is done in a document called a Statement of Defence. Since the proper preparation of the Statement of Defence presupposes that the defendant's solicitor possesses a thorough understanding of the case from his client's point of view, it is obvious that at some point between the serving of the Statement of Claim and the preparation of the Statement of Defence the lawyer who has been charged with your defence will want to see you and go over the case with you in detail.

He is going to want to learn from you every minute fact having any bearing on the action, and after he has questioned you for a few minutes you will begin to appreciate the extreme importance of the medical records to which we referred earlier. In all probability months have passed since you treated the patient in question and it is not much help to your lawyer to say "I think I did this" or "I think I did that". You should be able to say to him with absolute certainty that you did in fact do so-and-so, and my experience has been that a doctor who does not maintain adequate records cannot meet this test, thereby prejudicing his own defence. Another thing to remember is that when counsel first received your case he probably knew nothing whatever of the symptoms, treatment or medical problems involved, and yet by the time the action goes to trial he should be as conversant as possible with all of these matters. It is your responsibility during the first and subsequent interviews with your lawyer to teach him all that you possibly can about this particular field of medicine. You must be patient with him and above all you should not be content merely to sit back and answer the questions which he will put to you. It is up to you to do some thinking on your own and to mention to him any matter which may have a bearing on the case, regardless of how trivial it may appear to you. What may appear trivial and unimportant from a medical point of view may be of extreme importance from a legal standpoint. This is one of the things your lawyer must decide, but he cannot make such a decision unless you acquaint him with the medical facts. Even if your lawyer comes to the conclusion that much of the information which you have given him has no direct bearing on the case, nevertheless it all goes to give him a more complete and thorough understanding of the problem, and for this reason, if for no other, is extremely worthwhile in your own interests.

Following receipt of the Statement of Defence the plaintiff may, if he so desires, or feels it necessary in the particular circumstances, serve and file a "Reply", which is simply what the name would indicate. These documents, the Statement of Claim, the Statement of Defence and the Reply, constitute what are called the Pleadings and are the written record on which the action will be tried.

We now come to probably the most important pre-trial proceedings in the whole action, namely the Examination for Discovery. On the Examination for Discovery, counsel for the defendant may examine the plaintiff under oath and counsel for the plaintiff may examine the defendant under oath on the information, knowledge and belief of the respective parties with regard to all matters touching on the action in question. The questions and answers are taken down by a shorthand reporter and are subsequently transcribed and made available to each of the parties. Assuming that the Examination for Discovery is well conducted, each side will know at its conclusion precisely the facts on which the other side is relying and consequently will know the case which has to be met. Also the questions asked and the answers given by the defendant on his Examination for Discovery may be read into the record at the trial by the plaintiff if he so desires, thereby becoming part of the plaintiff's case at trial. The defendant cannot use his examination of the plaintiff in quite the same way, but he can use the transcript at the trial for purposes of cross-examination. It follows, of course, from what I have just said, that the answers given on the Examination for Discovery remain with you during the rest of the action and, while they may be contradicted or amended or denied at the trial, the very fact

that the same question or series of questions was answered in one way at one time and in another way at another time may tend to weaken materially the case as a whole and will at best cast doubt on your position with regard to that particular point. Therefore, before submitting to an examination for discovery, if you should ever find yourself in the position of having to do so, you should review matters thoroughly in your mind so that when the Examination takes place you can answer concisely and authoritatively any question which may be put to you, satisfied in your own mind that the answer you have given is one which you are prepared to support at any time and in any circumstances.

The time has finally arrived when we must prepare the action for trial. You will remember that in the beginning I quoted from an article by Mr. Haines outlining the standard of care expected of a physician. All the information now available to us must be examined with a view to determining whether or not the doctor in question met those standards of care in this particular case. Did he use a reasonable degree of learning and skill ordinarily possessed by physicians and surgeons in the locality in which he practises, or did he do something or omit to do something which other doctors would or would not have done? Was the judgment which he exercised in the particular case reasonable or unreasonable, having regard to all the medical information available to him at the time and examined again in the light of what comparable physicians would have done in the same circumstances? Was the technique which he used a modern, approved and accepted technique, generally used in the profession, or was it a technique which had been discarded by the profession many years ago? If he was a specialist, the burden of duty upon him is somewhat greater and the foregoing questions must be examined in relation to what other specialists in this field would have done. To ascertain the answers to these problems we must now approach other members of the profession and discuss the case with them in the same detail and with the same thoroughness as it has already been discussed with the defendant himself. Contrary to what many people think, it has been my experience that doctors do not automatically rally to the defence of another doctor when it is apparent that the defendant doctor has been guilty of carelessness or has departed from recognized and approved practices and principles. You must remember that the members of the Council of this Association are themselves practising physicians and surgeons highly specialized and qualified through experience and training in their own fields and if they themselves feel that they could not honestly stand up in Court and justify the actions of a defendant doctor, it can hardly be expected of them that they would request one of their fellow practitioners in another part of the country to do so. Personally, I have no quarrel whatever with this attitude, nor do I feel that the medical profession as a whole should or does think otherwise. If a doctor has been negligent and careless in the treatment of one of his patients, there seems to me to be no reason why the law of negligence should not be applicable to him in the same way as it is to any other individual. We have settled many cases out of court when it was our firm conviction that the action could not be defended and that we could neither ask nor expect the profession to support the actions of a particular doctor in a particular case. On the other hand, if it appears to your Council that the treatment prescribed and the manner in which such treatment was carried out was perfectly proper and recognized even though the result may have been unfortunate, we have no hesitation whatever in defending such a doctor to the limit and in requesting the support of the profession in his defence. I do not see that anyone can criticize either your Association or your profession as a whole for taking this stand, because I can think of no logical reason why a doctor should be deemed to be guilty of negligence or malpractice simply because he is a doctor.

The case is now ready for trial. In most provinces the action is tried by a judge alone, although there are some provinces where a jury is allowed in malpractice actions. The plaintiff puts in his case first through such witnesses as he may determine, including in many cases medical witnesses. Again I cannot object to the practice of doctors testifying on behalf of a plaintiff, nor do I feel that the medical profession should itself criticize any doctor who so testifies. The plaintiff's witnesses may, of course, be cross-examined by the defendant's counsel and when the plaintiff's case has been completed the same procedure is followed by the defendant. At the conclusion of the evidence, both counsel are entitled to sum up the evidence and to refer to the principles of law on which they rely. This concludes the trial, and while in some cases judgment may be delivered immediately from the bench, in most cases the judge prefers to reserve his decision in order to give himself an opportunity of reviewing the evidence which was adduced before him in the course of a trial which may have lasted a week or longer and also to consider the principles of law referred to him by counsel. In due course he will render judgment, finding for either the plaintiff or the defendant.

This concludes the conduct of a typical malpractice action, aside from an appeal, with which I do not propose to deal in this paper. Months and even years may have elapsed since the matters giving rise to the cause of action occurred, and even since the action itself was begun. During all this period many doctors who find themselves involved in such an action seem to feel that the sword of Damocles is hang-

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ing over their heads. While an action for malpractice is a serious thing and cannot be treated lightly, nevertheless it is a great mistake to allow it to assume proportions far beyond its importance. You must still carry on your practice and continue to give to your patients the service, attention and understanding which they expect of you and which you are capable and competent of fulfilling. As you know, I am not a doctor, but I am sure that if the situation were reversed and I consulted my physician his advice to me would be to carry on in my normal fashion and that the other matter would take care of itself in due course.

In closing there are two thoughts which I would like to leave with you.

In the first place I would like to commend to you the members of the Council of this Association. These men are all outstanding physicians and surgeons in their own particular fields, with large busy practices. Somehow they still find time to devote two evenings a month to the work of this Association and the interests of its members. I have never known them to refuse the most thorough consideration of any problem placed before them for decision, regardless of what hour in the evening it might be. If a problem is particularly urgent they are always ready to find time at the hospital or in their offices during the day to discuss the matter and offer their advice. Their conscientiousness, their willingness and their sincere interest in the welfare of this Association is something in which you as its members may take great pride.

In the second place, during the past year your Association has been concerned with somewhere in the neighbourhood of 60 to 100 cases. When you examine this in relation to the thousands and thousands of treatments prescribed and patients seen each day throughout the year, the figure becomes almost insignificant. At the same time a number of these cases could have been prevented by the exercise of ordinary care and common sense on the part of the doctor involved. This being so, the number of such cases can be reduced in the future by each doctor's refusing to allow himself to become even the slightest bit careless. An honest mistake in judgment is easily understandable. Carelessness cannot be justified either in the medical profession or in any other field. The reputation enjoyed by the medical profession in Canada and throughout the world is such that no member of the profession should lightly do anything that might destroy it. The difficulties of practice have undoubtedly increased tremendously over the last two or three decades, but in spite of these difficulties the reputation of your profession will be maintained and enhanced and the number of malpractice actions decline accordingly if all of its members continue to follow those sound and basic principles on which that reputation has been established.

MISLAID FOREIGN BODIES DURING SURGERY

III. INSTRUMENTS

J. H. B. HILTON, M.D.,* *Ottawa*

THE PREVIOUS two articles of this series have dealt with sponges and packing left in the wound after surgery. This article has to do with instruments left in the operation site and not missed until symptoms appeared later.

As illustrations, the following two cases are cited.

CASE 1

This woman had a Cæsarean section done by her surgeon, and at the same time a fibroid and her appendix were removed. The operation was entirely successful and she made an uneventful recovery. Some five years later she began to have some pelvic pain and was found to be running an irregular fever. Pelvic examination revealed infiltration and œdema in the left fornix. This subsided with penicillin administration. She was seen again seven months later, complaining of sacral pain. Examination at this time showed a hard linear mass in the left lower abdomen and fixed to the sacrum. She was suspected of having cancer and was sent to a cancer clinic. Here radiography showed a pair of artery forceps in the abdominal cavity. The patient had the forceps removed, sued the surgeon who carried out the original operation, and was awarded approximately $5,000 in damages and costs.

CASE 2

This woman was operated upon and her gallbladder removed. She did well and made a good recovery. She soon, however, began to have upper abdominal pain which came and went but did not incapacitate her. About three years after her operation she saw another doctor because of these complaints and he had some abdominal radiographs taken. These showed a curved artery forceps in the upper abdomen. The forceps was removed and the patient instituted action against the surgeon who had left the forceps. The case was settled for $3,000.

These two cases illustrate that instruments can be left behind in the wound and the surgeon be quite unaware that a serious accident has occurred. They also illustrate the fact that such mislaid instruments can remain in the abdominal cavity for long periods of time before symptoms of any severity appear. Although such accidents are fortunately rare, there appear to be more such cases coming to the Association's attention lately.

There are two possible ways of avoiding such accidents. One is by having an instrument count carried out in much the same way as sponge counts are done. Some hospitals have adopted this system. The other way is to have x-ray or fluoroscopic equipment in the operating room so that all patients can be screened by x-ray before leaving the room. If no such arrange-

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ments exist, the surgeon must be sure before he closes a wound that nothing has been left behind. If he is doubtful, he should carry out a painstaking search to make sure.

As it stands now, the surgeon is directly responsible for such mishaps as far as the courts are concerned. It is also true that when such an accident happens and suit is brought against the offending surgeon, damages are almost always awarded to the plaintiff. Such cases are almost indefensible in court, and damages for all actions seem to be getting higher.

To sum up this series of three articles, the surgeon must insist at every operation that a careful sponge count be done, a record of the count made and certified as correct. He must insist that all drains or packing be recorded. He must make sure that no instrument or other foreign bodies are left in the wound. Only by continuous care can the surgeon avoid unfortunate accidents of this kind.

SPECIAL CORRESPONDENCE

The London Letter

(from our own correspondent)

Lord Horder

The death of Lord Horder, on August 13, removes from our midst a great clinician and one of the few remaining "characters" in English medicine. There were few aspects of the national life with which he was not concerned. Family planning, cremation, noise abatement, industrial medicine, nutrition, rheumatism, cancer research—these are but a few of the activities with which he was actively associated during his long and honourable career. He was by no means the only leader of the profession who foresaw the stultifying effect of totalitarianism on the practice of medicine, but he was the only one who consistently opposed the more reprehensible features of the National Health Service Act at all stages. Throughout his long life, however—he was 84 when he died—he remained first and foremost a great clinician in the Sydenham tradition. He was indeed "a part of all that he met", and the country is the poorer for the passing of this distinguished son of Bart's; small in stature but great in courage, and the eloquent supporter of all measures for the amelioration of the lot of his fellow-citizens.

Clean Air Bill

The Government has shown commendable promptitude in introducing its promised Clean Air Bill to carry out the recommendations of the Committee on Air Pollution which, under the chairmanship of Sir Hugh Beaver, presented its report last year. The Bill prohibits the emission of "dark smoke", which is defined as smoke as dark as Shade 2 of the Ringelmann Chart, from any chimney. No new furnace (other than a domestic furnace) may be installed unless it is capable of being operated continuously without emitting any smoke. Users of existing furnaces (other than domestic furnaces) are required to take all practicable steps to minimize the emission of grit and dust. All new furnaces which burn pulverized fuel or solid fuel in large quantities are required to be fitted with equipment to arrest grit and dust. Local authorities are empowered, by Order, to designate "Smoke Control Areas". In such areas the emission of any kind of smoke from the chimney of any building will be prohibited. In such areas owners or occupiers of existing private dwellings are to receive grants toward the cost of adapting or replacing their grates or stoves, so as to enable smokeless fuel to be used. These grants will cover not less than 70% of such approved expenditure. No grant, however, will be payable in respect of houses built after the Bill comes into force. It looks as if at long last the powers that be realize that clean air is as important to health as clean water and clean food.

Subsidizing Litigiousness

The annual report of the Medical Defence Union has some outspoken comments on the working of the Legal Aid Scheme, under which individuals who are unable to pay their legal expenses can apply to a tribunal for a Legal Aid Certificate which allows them to obtain legal assistance free of charge. According to the treasurer of the Medical Defence Union, "The working of the Legal Aid Scheme continues to foment litigiousness and to encourage allegations, often baseless, against members of the medical and dental professions which would never have been made if their authors had known they were liable to have costs awarded against them. These cases cast heavy burdens on the Union's finances. Although the injustices to which the scheme not infrequently leads have been admitted and even deplored by the judiciary, there seems no prospect of any effective redress." A case in point is quoted in the Report, in which in giving judgment for the defendant, the Judge described the case as an abuse of the Legal Aid Scheme. He found that there had been no negligence or any semblance of negligence on the part of the doctor and said that even if the plaintiff had been able to satisfy him on this score the damages to which he would have been entitled total 3s. 8d. only. One of the injustices of the Scheme is that a successful defendant can rarely hope to receive costs from a plaintiff who has been assisted through the Scheme. In other words, as the Medical Defence Union points out, "A defendant, if successful, has nothing to gain except a public exoneration from blame for alleged negligence, while a plaintiff has nothing to lose by failure and everything to gain by a successful action."

It is only fair to point out that a distinguished Queen's Counsel has recently expressed the view that "there are no reliable figures showing to what extent any increase in medical litigation has been due to the Legal Aid Scheme". On the other hand, in his opinion, what "is beyond doubt is that the local committees who are charged with the duty of issuing Legal Aid certificates have been doing their work well, that is to say, taking considerable care to satisfy themselves that a prima facie case exists before issuing a Legal Aid Certificate".

Epidemic at Royal Free Hospital

The epidemic which has closed the Royal Free Hospital, and to which reference was made in this Correspondence last month, has now passed its peak. Over 100 members of the staff have been involved. As the epidemic progressed, however, considerable doubt was thrown on the original diagnosis of "glandular fever". Indeed, at the time of writing, an official statement has been issued by the Hospital authorities to the effect that "it is now difficult to sustain this, or any other, diagnosis". Happily the disease has run a benign course in most cases. The predominant feature of the epidemic has been involvement of the lymph nodes, liver and spleen, with signs and symptoms of encephalomyelitis or polyneuritis. A peculiar feature of the infection has been the high incidence of vertigo. The Paul-Bunnell test has been consistently negative. Even when the infection has been severe, pyrexia has been slight or absent. The current hypothesis is that it is a virus infection, and active investigations are being made from this point of view.

London, September 1955. William A. R. Thomson

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ABSTRACTS *from current literature*

MEDICINE

Apical Diastolic Murmurs Simulating Mitral Stenosis.

II. Graphic Differentiation.

LUISADA, A. A., HARING, O. M. AND ZILLI, A. B.: ANN. INT. MED., 42: 644, 1955.

It has long been known that patients with a normal mitral orifice may have a low-pitched diastolic or presystolic murmur indistinguishable upon auscultation from that of mitral stenosis. Such a murmur has been described in aortic regurgitation, pericarditis, hypertensive heart disease, myocarditis and rheumatic heart disease without mitral stenosis.

In this investigation, 34 patients were studied, with the following criteria: (1) They had a low-pitched diastolic rumble, a presystolic murmur, or both. (2) The "functional" nature of the murmur was demonstrated by autopsy, surgery or complete disappearance of the murmur following improvement in or cure of the clinical condition. Clinical, roentgenological, electrocardiographic and phonocardiographic studies were carried out in all patients.

Certain positive data were found helpful in recognizing the nature of the murmur: the murmur is frequently loud and occurs later in diastole; it is often recorded over a large area of the chest, and there is frequently a third sound.

Certain negative data were also found helpful: there is no opening snap of the mitral valve; the main vibration of the first sound has a normal relationship with the QRS complex, when the electrocardiogram and phonocardiogram are superimposed.

The authors discuss the mechanism of production of these apical diastolic murmurs which simulate mitral stenosis, and conclude that phonographic examination results in recognition of the "functional" nature of the murmur in a great majority of cases. S. J. SHANE

Diagnosis of Cardiac Hypertrophy in the Aged: Clinical Pathological Correlation in 55 Individuals.

RODSTEIN, M.: AM. J. M. SC., 229: 525, 1955.

In an effort to assess the accuracy of clinical methods of detecting cardiac enlargement or hypertrophy in elderly patients, the findings on physical examination, chest film and electrocardiogram in 55 men and women ranging in age from 63 to 95 years have been correlated with observations at necropsy.

The diagnosis of cardiac enlargement or hypertrophy was made correctly in 32 cases, all found to have moderate to marked hypertrophy of the ventricular muscle at post-mortem examination. Slight hypertrophy of the ventricular muscle was demonstrated in seven cases at post-mortem examination but was not detected by any of the three clinical methods of examination. In 16 cases without any hypertrophy of the ventricular muscle at post-mortem examination, six were diagnosed as showing cardiac enlargement on roentgenogram and one on physical examination. Cardiac dilatation was considered as the probable cause of these diagnoses. None of this group manifested any of the electrocardiographic changes of cardiac hypertrophy.

Of the 32 cases with moderate or marked hypertrophy of the ventricular muscle at necropsy, 20 were thought to show enlargement on physical examination. Nineteen of 24 with available roentgenograms were diagnosed as showing enlargement on chest film. Nineteen of 31 with available electrocardiograms were thought to show cardiac hypertrophy by electrocardiographic criteria. In all, enlargement or hypertrophy was diagnosed by one or more of the three methods.

The most sensitive electrocardiographic criteria for hypertrophy were RaVL greater than 11 mm. and Rl + S3 = 25 mm. or greater. This was attributed to the increased electrically horizontal position and counterclockwise rotation of the heart associated with aging.

There was no consistent pattern of co-existence of diagnoses of enlargement or hypertrophy by the three methods in the cases with moderate or marked hypertrophy of the ventricular muscle at post mortem.

In cases with moderate or marked hypertrophy at post-mortem examination, diagnoses of enlargement by roentgenogram and physical examination tended to occur more frequently in the same individuals than of hypertrophy by electrocardiographic criteria. This was attributed to the fact that the former are measures of size, the latter of hypertrophy.

In cases with moderate or marked ventricular hypertrophy at necropsy, individuals with enlargement on roentgenogram and no positive electrocardiographic criteria of hypertrophy were commoner than the reverse. This was attributed to the effect of increased incidence of coronary and myocardial disease in this age group in diminishing the accuracy of the electrocardiographic criteria of hypertrophy. S. J. SHANE

Duodenal Ulcer as an Occasional Etiologic Factor in Obstructive Jaundice.

SNAPE, W. J.: ANN. INT. MED., 42: 1001, 1955.

Duodenal ulcer may be an etiological factor in certain cases of obstructive jaundice. The regional anatomy of the area adjacent to the duodenum suggests that the duodenal ulcer may cause occlusion of the common duct by the following means: first, the ulcer, or its inflammatory reaction, may impinge upon the biliary papilla; this is probably an infrequent mode of obstruction. Second, since in 75% of individuals the common duct travels behind the superior portion of the duodenum and is embedded in the pancreas, inflammation and œdema of the pancreas resulting from a posterior penetrating ulcer could compress the common duct in this region. Finally, deeply penetrating or perforating ulcers may involve the gastrohepatic ligament and thereby cause obstruction in the more proximal portions of the common bile duct.

Case reports on six patients are presented; in three the diagnosis was confirmed at exploration, one bled from his ulcer and was jaundiced, and one had two recurrences, with liver biopsy in each instance showing evidence of obstructive phenomena associated with negative cholecystograms.

The author feels that duodenal ulcer should be suspected as an etiological factor in cases of obstructive jaundice which cannot be otherwise explained.

S. J. SHANE

Sources of Upper Gastrointestinal Bleeding in Liver Cirrhosis.

DAGRADI, A., SANDERS, D. AND STEMPIEN, S. J.: ANN. INT. MED., 42: 852, 1955.

It is self-evident that an accurate determination of the source of bleeding in liver cirrhosis is important for diagnosis and therapy. Much too often it is assumed that a patient who bleeds and has liver cirrhosis is bleeding from œsophageal varices. One striking example of such an erroneous assumption is reported in this paper, in a case in which the emergency management was entirely confined to a tamponade of the lower œsophagus and cardia without due attention to accurate diagnosis. In this particular case, the post-mortem examination disclosed that the bleeding came from a benign gastric ulcer. It is therefore important that, in a patient who

bleeds and has liver cirrhosis, *all* of the causes of gastrointestinal bleeding should be considered.

In a study of this problem, involving 121 patients with hepatic cirrhosis, and controlled by endoscopy and needle biopsy of the liver, it was found that the commonest sources of upper gastrointestinal bleeding in hepatic cirrhosis are œsophageal varices, hæmorrhagic gastritis and duodenal ulcer. In contrast to generally accepted views on the subject, hæmorrhagic gastritis ranks almost equally with varices as a frequent cause of bleeding in cirrhosis of the liver.

The authors conclude that œsophagoscopy and gastroscopy are important adjuncts in the diagnostic study of upper gastrointestinal hæmorrhage in cirrhosis of the liver.

S. J. SHANE

Tuberculomas of the Central Nervous System: Four Cases Successfully Managed with Surgery and Chemotherapy.

SCHWARTZ, M. ET AL.: ANN. INT. MED., 42: 1076, 1955.

The writers present four cases to exemplify the successful management of tuberculomas of the central nervous system by surgery and antimicrobial therapy. All patients presented the clinical findings of a tumour of the central nervous system, later shown to be a tuberculoma. Surgical treatment was that for extirpation of any central nervous system tumour, but all patients were treated postoperatively with streptomycin, PAS and isoniazid.

The writers stress the importance of treating tuberculomas of the central nervous system postoperatively as though tuberculous meningitis were present. They also feel that, in any case of meningitis in which a tuberculous etiology is strongly suspected or proved, further investigative procedures such as pneumoencephalography might be indicated when the meningitis has resolved. The purpose of this would be to determine the presence of an underlying tuberculoma, which might have caused the meningitis by eroding into the subarachnoid space, and which might be amenable to surgical extirpation. Such a sequence may account for relapses in apparently adequately treated cases of tuberculous meningitis. Four illustrative cases are presented, including an unusual case of spinal epidural tuberculoma.

S. J. SHANE

SURGERY

Cholangiography.

KANTOR, H. G., EVANS, J. A. AND GLENN, F.: A. M. A. ARCH. SURG., 70: 237, 1955.

A plea is made for the more frequent use of cholangiography on the operating table. In the New York Hospital indications for common duct exploration during cholecystectomy are recognized as: (1) palpable stone in common duct; (2) history of jaundice with pain or fever; (3) thickened common duct; (4) contracted gallbladder containing stones; (5) dilated cystic duct; (6) enlarged head of pancreas; (7) prolonged history of biliary disease in a patient over 60 years old.

A study was made of 171 operative cholangiograms. Normal variations are discussed, especially those of the distal end of the common duct and the junction of the cystic and common ducts, and air bubbles in the dye. The appearance of stones, air bubbles, strictures and neoplasms is described.

In the series examined 24% showed abnormalities in the cholangiograms: 40 showed stones, 8 tumours, 6 strictures and one choledochal cyst. Only 15 of the 40 stones were causing obstruction. The reasons for unsatisfactory examinations are discussed.

BURNS PLEWES

Five-year Survivors Following Pancreato-spleno-total Gastrectomy for "Advanced" Cancer of the Stomach.

BRUNSCHWIG, A.: ANN. SURG., 141: 62, 1955.

Four cases of cancer of the stomach were operated upon with long survival. The whole stomach, body and tail of the pancreas, spleen and the adjacent omenta and posterior lymphatic-bearing tissue were removed. One patient with 18 months' history and a weight loss of 25 lb. had reticulum-cell sarcoma and is living and well at 6 years, 4 months. A second patient with adenocarcinoma of the antrum infiltrating the gastro-colic omentum and lymph nodes seemed to have a recurrence apparently controlled by x-ray therapy and is well 5 years, 1 month after operation. The third case had been declared inoperable elsewhere after laparotomy because of posterior fixation. Resection en masse was done. The adenocarcinoma of the stomach showed squamous metaplasia and the adhesions to the pancreas were proved inflammatory. He is well though alcoholic, and working after five years. The fourth patient had dyspepsia for five years before laparotomy elsewhere led to a diagnosis of inoperable carcinoma at age 39. After resection (which included the adrenal) he went home in 13 days, remained well except for occasional regurgitation, and is working hard two years later.

Whether the radiotherapy two of these patients received was important to their survival is discussed. It seems impossible to decide at operation how extensively the cancer has involved other organs. The number of failures of the extensive procedure is not reported.

BURNS PLEWES

Surgical Treatment of Diverticulitis of Colon.

GILCHRIST, R. K. AND ECONOMOU, S.: A. M. A. ARCH. SURG., 70: 276, 1955.

Though one-third of all people over 45 years old have diverticula of the colon, only a few require surgical intervention. These pulsion diverticula are thin walled and may perforate with only moderately increased pressure within the colon as from an enema, or obstruction from a carcinoma.

At the Presbyterian Hospital in Chicago, the indications for operation in diverticulitis are as follows: free perforation is treated by obstructive resection or closure and transverse colostomy; abscesses are drained and usually the diseased bowel is resected later; recurrent and disabling bouts of diverticulitis are indication for resection, as are rectovesical fistulæ or repeated bouts of cystitis and pyelitis, failure to exclude carcinoma, and bleeding with diverticulitis. The ideal operation is resection of the descending and sigmoid colon to the peritoneal reflection with end-to-end anastomosis.

BURNS PLEWES

The Surgical Treatment of Aortic Insufficiency.

BAILEY, C. P. AND LIKOFF, W.: ANN. INT. MED., 42: 388, 1955.

These workers have used surgery to correct aortic insufficiency in patients seriously ill with a dynamic lesion. Various methods were used, including the Hufnagel valve, flat "tailed" discs of silicone rubber, and other forms of prostheses. No method has yet been devised to correct aortic insufficiency without the use of prosthetic materials. The article outlines the etiology, clinical manifestations, pathology and surgical techniques used in the correction of aortic insufficiency in seven patients. With the use of nylon and silicone rubber prostheses, the authors' surgical efforts were rewarded by five survivals, with two deaths. Four of the survivors have shown objective evidence of benefit, with elevation of the diastolic blood pressure, characteristic changes in the

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arterial pulse wave, and diminution of other characteristics of the disease.

The authors also describe their experiences with the technique of surgical constriction of the aortic root close to the annulus, in an effort to decrease the diameter of the aortic ring and thus indirectly increase the efficiency of the aortic cusps. This technique has been applied in five cases, with two deaths and two clinical improvements. This is a pioneering effort only, and it is still considered unwise to subject any patient to the rigours and risks of this new technique unless he is already reduced to the terminal phase of his disease without possibility of medical salvation.

Nevertheless, the rather remarkable and immediate improvement obtained in some cases suggests that the authors are at last on the right course in the treatment of this crippling disease. S. J. SHANE

Surgical Management of Massive Acute Upper Gastrointestinal Hæmorrhage.

WELCH, C. E., ALLEN, A. W. AND DONALDSON, G. A.: NEW ENGLAND J. MED., 252: 921, 1955.

[This article is an important contribution to a complex problem and warrants perusal in the original. It is difficult to abstract it without distortion.—N.S.S.] The authors found the mortality from massive upper gastrointestinal hæmorrhage at the Massachusetts General Hospital to be 13% for the periods 1923 to 1932 and 1938 to 1947. The present series of 269 cases covers the period 1948 to 1953. The mortality rate had risen to 17%. This increased mortality is not believed due to the marked increase in incidence of surgical therapy (operation being carried out in 69%), but is ascribed to the average increase in age of these patients over former series, and to postoperative complications.

Emergency surgery is indicated in the presence of massive upper gastrointestinal bleeding when the bleeding is persistent or recurrent. X-ray examination should be carried out on these patients to determine the cause of the hæmorrhage; if it is due to peptic ulcer, operation should be carried out at once. Spontaneous cessation of hæmorrhage is much more likely if it is due to gastritis or hiatus hernia, where a more conservative attitude may be adopted.

Technical details of anæsthesia, operation and postoperative care are of vital importance and are discussed in this paper. NORMAN S. SKINNER

Relation of Gallstone Disease to Angina Pectoris.

RAVDIN, I. S. *et al.*: A. M. A. ARCH. SURG., 70: 333, 1955.

The association between gallstone disease and cardiac disease has been observed for nearly half a century. It has been shown that if the vagi are intact, distension of the common bile duct lowers myocardial blood flow. A series of cases with clinical and electrocardiographic evidence of coronary disease and of gallstones illustrates the argument that with co-operation between surgeon and internist such patients are greatly improved by surgical treatment of the gallbladder and/or common bile duct stones. The diagnosis of angina should not be regarded as a contraindication to an operation on a diseased biliary tract unless the heart disease is so far advanced that the operative hazard outweighs the chance of benefit.

A review of such a group of patients at the Hospital of the University of Pennsylvania shows an operative mortality rate of 3.8%. In the 1922-36 period the mortality was 16.2%, and from 1937-44 it was 2.7%. In 106 patients, from 1944-47, there were no deaths. The risk of operations for gallstones in patients with heart disease is not as great as many clinicians believe.

BURNS PLEWES

Effect of Intravenous Calcium Gluconate on Post-Transfusion Hypotension.

STRAWITZ, J. G., HOWARD, J. M. AND ARTZ, C. P.: A. M. A. ARCH. SURG., 70: 233, 1955.

Observations on severely injured casualties in Korea showed a post-transfusion hypotension which often responded to the intravenous injection of calcium gluconate. The large amount of citrate received with the blood is thought to bind calcium, and the calcium gluconate to assist in the return of calcium to the ionized state. The rise in pulse volume and blood pressure was often striking, and respiratory stimulation may also be a factor in increasing cardiac output. The theory is not yet proved. BURNS PLEWES

Occult Carcinoma of Breast.

GERSHON-COHEN, J., INGLEBY, H. AND HERMEL, M. B.: A. M. A. ARCH. SURG., 70: 385, 1955.

Occult carcinoma of the breast usually means that some remote metastatic lesion such as an enlarged axillary lymph node or tender vertebra is the only evidence of disease. The primary lesion is undetected clinically. Roentgenography of the breast may disclose malignant lesions only a few millimetres in diameter. Five such cases are described. These occurred in 175 successive cases of carcinoma of the breast.

X-ray examination may also aid in the diagnosis of malignant breast disease thought to be benign clinically. BURNS PLEWES

OBSTETRICS AND GYNÆCOLOGY

The Treatment of Hydramnios Complicated by Anencephaly.

RUSSELL, C. S. AND ABBAS, T. M.: J. OBST. & GYNÆC. BRIT. EMP., 61: 610, 1954.

As a working rule, the association of anencephaly and hydramnios should be treated by artificial rupture of the membranes as soon as a diagnosis is made, with the possible exception of cases diagnosed before the 32nd week of pregnancy. ROSS MITCHELL

Incision of the Cervix.

COPE, E.: J. OBST. & GYNÆC. BRIT. EMP., 72: 432, 1955.

The operation of incision of the cervix has a real but strictly limited use. The operation is indicated when the greatest diameter of the head has descended below the cervico-vaginal junction and early delivery is required, the only obstacle being the undilated cervix.

Thirteen cases of cervicotomy without maternal or fetal mortality are described. The hazards and mechanics of cervicotomy are considered, and an attempt is made to define the use and limitations of the operation. The technique is described. ROSS MITCHELL

Accidental Hæmorrhage.

BARRY, A. P., FEENEY, J. K. AND GEOGHEGAN, F. J.: BRIT. M. J., 2: 12, 1955.

Hæmorrhage, renal failure and fetal asphyxia render accidental hæmorrhage a serious or potentially serious condition. In the severe case of accidental hæmorrhage the bleeding is of a special type—that is, due to a fall in blood fibrinogen. The clinical manifestations of this clotting defect are described. The pathogenesis of anuric renal failure is presented in brief.

A scheme of treatment for the mild, moderate and severe case of accidental hæmorrhage is suggested. In Dublin, over the past two years, the results of this treatment have been most encouraging.

Notes relating to splanchnic block and to Schneider and Shea tests for fibrinogen deficiency are included.

A summary of the statistics of accidental hæmorrhage in Dublin over the 25-year period 1929-53 is presented.

ROSS MITCHELL

PÆDIATRICS

Successful Treatment of Beta Hæmolytic Streptococcal Infections in Children with a Single Injection of Repository Penicillin (Benzathine Penicillin G).

BREESE, B. B. AND DISNEY, F. A.: PEDIAT., 15: 516, 1955.

In 1,175 proven cases of beta hæmolytic streptococcal infections in children, treatment consisted of a single dose of 600,000 units of benzathine penicillin G intramuscularly. If a case appeared clinically to be one of streptococcal infection, treatment was begun at once, without awaiting the result of cultures.

Response was prompt and excellent, and no severe reactions occurred. Usually the patient was non-infectious in 24 hours. Complete cure was obtained in 94%. Recurrences (any illness within 61 days, accompanied by positive throat culture) numbered 118, but 88 occurred in the latter half of this period, and were probably reinfections. A carrier state was present at follow-up examination in 35 cases.

This treatment appeared to prevent suppurative complications, rheumatic fever and nephritis. In cases with deep-seated lesions, the authors prefer to use repeated doses of penicillin preparations which give higher concentrations in blood and tissues.

I. J. PATTON

Chlorpromazine in the Control of Vomiting in Children.

DAESCHNER, C. W. ET AL.: AM. J. DIS. CHILD., 89: 525, 1955.

A preliminary clinical evaluation of chlorpromazine in the symptomatic treatment of 100 cases of vomiting in infancy and childhood indicates that it is both safe and effective. In 83 cases, the response was good or excellent, and parenteral fluids were not required. A transitory sedative effect was observed in most of the successfully treated cases.

The failures were in two cases of pyloric stenosis, requiring operation; one case of achalasia of the intestine with superimposed measles encephalitis; two cases of bulbar poliomyelitis; one case of severe laryngotracheobronchitis with paroxysmal cough; one case of chronic vomiting and extreme malnutrition, of unknown origin; one case of non-specific gastroenteritis. In the remaining nine cases, the response to chlorpromazine was not definite, and parenteral fluids were needed.

Toxicity was not significant in this series, but it is recommended that possible complications, such as agranulocytosis or liver damage, be kept in mind. When possible, the cause of vomiting should be determined before beginning treatment.

Effective dosage appears to be: intramuscular, 0.5 mg. per kg.; oral, 1.0 mg. per kg.; as suppositories, 2 to 3 mg. per kg.

I. J. PATTON

Therapy of Acute Rheumatic Fever.

DONE, A. K. ET AL.: PEDIAT., 15: 522, 1955.

A study was made of 62 children with acute rheumatic fever and without a previous history of rheumatic fever or cardiac murmur, treated either with ACTH, usually in a dosage of 1 i.u. per lb. per day, plus 500 mg. of ascorbic acid daily to aid its utilization (32 patients); or cortisone, usually in a dosage of 2.5 to 3.0 mg. per lb. per day (14 patients); or salicylates, usually 1 grain per lb. per day, in divided doses (21 patients); or bed rest only (13 patients). A group of 18, previously treated with ACTH, was re-examined.

The type of treatment had little effect on the incidence of major and minor manifestations of rheumatic fever. The only patient without carditis was in the "bed rest" group. All of the drugs produced prompt symptomatic response, joint symptoms yielding more rapidly to the hormones than to the salicylates. The erythrocyte sedimentation rate returned to normal much more quickly in the hormone-treated groups. All patients received antibiotic or sulfonamide prophylaxis.

At subsequent examinations over periods as long as 3¾ years, cardiac murmurs were rare in the hormone-treated groups, as compared to the other two.

For good results, dosage and duration of therapy must be individualized, having regard to body size, severity of illness, and response to treatment.

I. J. PATTON

THERAPEUTICS

Treatment of Acute Infectious Hepatitis. Controlled Studies of Effects of Diet, Rest, and Physical Reconditioning on Acute Course and on Incidence of Relapses and Residual Abnormalities.

CHALMERS, T. C. ET AL.: J. CLIN. INVEST., 34: 1163, 1955.

Based on careful clinical studies in military personnel, recommendations were drawn up for revision of the treatment of acute infectious hepatitis in the Armed Forces. They embody a distinct change in the presently accepted rest regimen and little or no change in diet. Their applicability to the more severe forms of the disease in older or less well-nourished patients has not been established.

Since physical activity to the point of fatigue may be harmful, patients with acute hepatitis should be hospitalized as soon as the diagnosis is made. They should be urged to rest in bed as long as acute symptoms persist. Once patients begin to feel well, regardless of the degree of jaundice, they should not be forced to stay in bed more than one hour after each meal. Restriction to the hospital ward, however, is essential to decrease undue activity or exertion. Activity on the ward should be allowed *ad lib.*; this circumvents the usual delay necessary for rehabilitation and appreciably shortens the period of hospitalization.

Patients treated in this way may be discharged from the hospital and physical reconditioning may be undertaken after the total serum bilirubin concentration is below 1.5 mg. per 100 ml. and the bromsulphalein retention in 45 minutes below 6% for a period of not less than one week. Patients whose bromsulphalein retention stabilizes between 5 and 10% may be discharged from hospital with safety. Those with persistently higher levels will require individual management.

After discharge all patients should be followed up for two weeks with weekly physical examinations, serum bilirubin determinations and bromsulphalein tests. Recurrence of abnormalities is rare and probably an indication for rehospitalization.

The optimal diet consists of about 3,000 calories, containing approximately 150 g. proteins and 150 g. fats. Intakes above this level should be *ad lib.* Although fried and greasy foods may cause indigestion, the fat contained in meat, eggs, and dairy products is not harmful and adds greatly to the palatability of the diet. During the stage of severe anorexia, the patient should be

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urged to take frequent small feedings. Intravenous glucose solution should be administered when necessary to maintain minimal caloric intake. Although the forcing of a high-protein, high-fat diet, by stomach tube if necessary, has been demonstrated to hasten recovery on the average, critically ill patients with fulminating disease or impending hepatic coma may be harmed by excess dietary protein. In these cases, therefore, it is unwise to administer more than a maintenance quantity of protein.

Intravenous protein hydrolysates, plasma or blood transfusions have no place in therapy of uncomplicated infectious hepatitis. B. L. FRANK

Cortisone in the Treatment of Infectious Hepatitis.

HUBER, T. E. AND WILEY, A. T.: ANN. INT. MED., 42: 1011, 1955.

The writers present the data assembled from a 17-month study, under controlled conditions, of over 200 cases of infectious hepatitis in the Army Hospital in Yokohama, Japan. Excluding possible serum hepatitis and other liver diseases, and using as a base line an icterus index of 25 units, they divided the patients into two groups, one on cortisone, and one as a control, both alternately in oral and parenteral form. Cortisone therapy resulted in a more rapid clearing of jaundice as measured by the average icterus index and serum bilirubin levels, than occurred in the controls. There was also a more rapid return of the appetite than in controls. The cortisone-treated patients consumed more food than did the controls, and there was also a greater gain in weight. The results of bromsulphalein tests in the cortisone-treated patients, on the average, returned to normal faster than they did in the controls, and patients treated with cortisone became clinically well more rapidly than did the controls.

Because of these findings, the writers feel that cortisone is of definite value in the treatment of hepatitis. They do not recommend the use of so potent a drug in the routine treatment of infectious hepatitis, but they feel that it should be of great value in patients not responding to conventional treatment. S. J. SHANE

Corticotropin (ACTH) in Heart Disease: Its Paradoxical Effect on Sodium Excretion in Resistant Congestive Failure.

CAMARA, A. A. AND SCHEMM, F. R.: CIRCULATION, 11: 702, 1955.

It has been increasingly realized that a number of œdematous patients fail to respond to the usual diuretic measures now in use, and truly resistant œdema is common. In these cases, diuretic measures, including vigorous use of mercurials, are of no avail, or may cause sodium excretion without weight loss or even a weight gain.

These workers have carried out a clinical trial of corticotropin, with detailed studies, since the spring of 1952, in apparently hopeless and terminal cardiac conditions with truly resistant œdema. The corticotropin was given intramuscularly in doses of 10 to 25 mg. (depending on body weight) every six hours for 10 to 12 days. Two patients received corticotropin intravenously. Careful electrolyte balance studies and eosinophil counts were done.

Of the 21 cardiac patients studied and treated, 17 (81%) were definitely benefited by the hormone. The benefits included: (1) Spontaneous diuresis occurring *during* treatment (usually on the 4th to 6th day, and continuing after the course of corticotropin therapy with loss of previously resistant œdema. (2) Diuresis occurring spontaneously after discontinuation of the hormone with loss of weight and œdema, and relief of intractable pulmonary congestion. (3) An altered response to mercurial diuretics, the patients now responding well. This change occurred either during or after the course of corticotropin therapy.

The authors suggest that this effect may be due either to some still-undiscovered adrenal hormone, produced in response to corticotropin stimulation, or that corticotropin may directly, or through some other adrenocortical hormone, improve the condition of the failing myocardium. They recommend further clinical trials of corticotropin in such patients. S. J. SHANE

INDUSTRIAL MEDICINE

Health Hazards of Drifting Parathion Dust Cloud.

BRAID, P. E., WINDISH, J. P. AND ROSS, C. R.: A. M. A. ARCH. INDUST. HEALTH, 11: 403, 1955.

Although parathion has been used for several years in liquid insecticides, in orchards, it has to date had only limited application as a dry dusting agent in Canadian horticultural operations. The authors, reporting from the Occupational Health Laboratory, Department of National Health and Welfare, Ottawa, describe a series of experiments designed to estimate some of the hazards likely to be encountered should this insecticide be widely used in the dusting of small crops. The dust may drift to adjoining fields and present a direct inhalation danger to workers there, and by deposition may render crops in those fields unsafe for handling or marketing. An assessment of these hazards is made by sampling the airborne dust and also the vegetation at distances up to 400 feet from the dusting machine.

A commercial insecticide consisting of 1% parathion in attapulgus clay was spread at 40 lb. to the acre with wind speeds ranging from 3 to 8 m.p.h. On the day of dusting, using the threshold limit proposed by the American Conference of Governmental Industrial Hygienists, it was found that the concentration of parathion in the air at breathing height was sufficient to constitute a respiratory hazard up to a distance of 260 feet from the source of propagation. A formula is given for calculating the limit for safe handling of vegetation, related to the dusting time and the distance from the source of dusting. In view of the toxicity of parathion, and the fact that several cases of poisoning following its use in liquid insecticides have been noted, the data given in this paper should prove most useful to those responsible for proposing control measures when it is used in its dry state. J. D. MEDHURST

Parathion Spray Concentrations and Residues in Quebec Apple Orchards.

BRAID, P. E., WINDISH, J. P. AND ROSS, C. R.: A. M. A. ARCH. INDUST. HEALTH, 11: 408, 1955.

A further report from the authors' combined study of parathion exposure and cholinesterase response of Quebec orchard workers is given in this paper. Spray mist samples were taken from the tractor-operator's breathing zone and subsequent leaf and residual air samples have also been examined.

Data are given on air concentrations and decay rates of the insecticide under various working conditions. Attention is directed to the importance of maintaining constant vigilance in handling parathion. Provided simple protective measures are observed, no toxic symptoms are likely to develop. Several cases of parathion poisoning which occurred in the area have been investigated and were found to be due to: (a) direct skin exposure during nozzle cleaning; (b) wearing of non-waterproof clothing drenched with spray; and (c) excessive skin contact with the insecticide during tank filling. J. D. MEDHURST

OBITUARIES

DR. IVAN CARMEN CLENDINNEN, a veteran army medical officer and a well-known private practitioner of Hamilton, Ont., died in that city on September 6. He was 56.

Dr. Clendinnen, who was born in Newborough, Ont., served as an infantryman in the First World War before studying medicine at McGill University, from which he graduated in 1924. He had been in practice in Hamilton since 1929, with the exception of five years during World War II. He joined the 5th Field Ambulance after setting up practice in Hamilton and was its commanding officer in 1939. In that year he went overseas, and in the next five years he commanded the 23rd Field Ambulance and the 2nd Field Dressing Station. He returned to Canada in 1944 to become chief instructor at the R.C.A.M.C. school at Camp Borden. He resumed command of the 5th Field Ambulance (Reserve) on his return to Hamilton in 1945 and retired as its commander in 1954.

Dr. Clendinnen is survived by his widow, a daughter, and a son.

DR. S. M. DABROWSKI, 44, died August 24 of injuries sustained in a highway accident involving the car in which he was accompanying a child to hospital. He died aboard a plane sent to take him to hospital in Edmonton.

Dr. Dabrowski was born in Paris of Polish parents and received his medical education in Poland. During the Second World War he was captured and held prisoner in Germany till 1945. He arrived in Edmonton in 1951, completed his medical examinations in Winnipeg, and began practice in Grimshaw, Alta., where he had recently moved from Edmonton. He is survived by his widow and sister.

DR. ANDREW GRAY, 82, of Chippawa, Ont., died in the Toronto General Hospital on September 18 as a result of injuries sustained when he fell from a tree while picking fruit.

Dr. Gray was born in Niagara Falls, Ont., and was a graduate of the University of Toronto. He had practised in Niagara Falls for a few years before moving to Chippawa, where he had been in continuous practice for 58 years.

He is survived by his widow and a sister.

DR. ALBERT HEYNINX of Montreal, an ear, nose and throat specialist and former physician to the Belgian royal family, died August 30 at the Montreal Neurological Institute of a cerebral hæmorrhage. He was 78.

A native of Ypres, Dr. Heyninx came to Canada from Belgium seven years ago, and had been a Canadian citizen for one year before his death.

A recipient of several awards from the Brussels court for his medical skill, he was chevalier and officer of the Order of Leopold, chavalier of l'ordre de la Couronne, and was awarded the medal commemorating the 100th anniversary of Belgium's independence. Dr. Heyninx was also an honorary professor at the University of Brussels.

He is survived by his widow.

DR. CLARENCE A. HOWARD, of Kingston, Ont., died at his home on September 8 after a short illness. He was 68.

Dr. Howard was born near Athens, Ont., and graduated in medicine from Queen's University in 1912. In addition to carrying on a general practice, he was for many years surgeon-in-chief at the Hôtel-Dieu Hospital and surgeon at Kingston Penitentiary.

Dr. Howard is survived by his widow and two daughters.

DR. DOUGALD G. JAMIESON, of Newfoundland, honoured with Senior Membership in the Canadian Medical Association at the June 1955 annual general meeting, died August 16 at the Grace Hospital, St. John's, at the age of 72.

Dr. Jamieson was born in Beaverton, Ont. He received his medical degree from the University of Toronto and interned at the Toronto General Hospital for two years. Persuaded by the late Dr. Wilfred Grenfell to begin his medical practice in Newfoundland where there was a need for well-qualified physicians, he established himself in the community of Wesleyville, and later Greenspond which he served for 15 years. Dr. Jamieson had been living since 1928 in St. John's, where he was a member of the staff of Grace Hospital, and where he specialized in dermatology and anæsthesiology. He is survived by his widow, a son and three daughters.

DR. ROBERT AVERY McCOMB died at his home in Toronto on September 11. He had served at the Toronto General Hospital for 30 years until 1951, when he retired to private practice.

Dr. McComb was born in Consecon, Prince Edward County, Ont. He graduated in medicine from the University of Toronto in 1904 and carried on a general practice in both Millbank and South River, Ont., for 10 years. He then took further training at the Toronto General Hospital and went from there to the Brady Institute at Johns Hopkins Hospital for postgraduate training in surgery. On his return to Canada he was appointed to the staff of the Toronto General Hospital. Until his retirement four years ago, he also taught in the department of surgery at the University of Toronto. He was a Fellow of the Royal College of Surgeons of Canada and of the American College of Surgeons.

Dr. McComb is survived by his widow, a son, and a daughter.

DR. JOSEPH MEGAS, who had practised in Edmonton for the past 20 years, died in that city on September 6 at the age of 73.

A native of Poland, Dr. Megas came to North America at the turn of the century and lived in Pennsylvania for five years before moving to Manitoba. Fifteen years later he entered medicine at the University of Alberta, graduating in 1926.

Dr. Megas is survived by his widow and three sons, one of whom is Dr. Constant Megas of Edmonton.

DR. JOHN ROBERT SPIER, retired medical practitioner of Montreal and North Hatley, died on August 21 in the Sherbrooke Hospital. He was a graduate of McGill University and had practised his profession in Montreal until 1930, when he moved to North Hatley. He had been living in retirement in Sherbrooke for the last decade.

Dr. Spier is survived by his widow and two daughters.

DR. ERNEST ARGYLE WHITE, 85, a practitioner for more than 50 years, died in Fenelon Falls, Ont., on August 28. Dr. White was born in Toronto. After graduating from the University of Toronto in 1895, he practised at Kinmount, Ont., for many years, before moving to Fenelon Falls, where he maintained an office for 30 years. He retired in 1950.

Dr. White is survived by a son, Dr. C. A. White of Long Branch, Ont., and by a daughter.

DR. JOHN THOMAS WHITE of Fort William, Ont., died suddenly at St. Michael's Hospital, Toronto, on July 29, at the age of 39.

Dr. White, who was born in Brussels, Ont., was well known as an athlete. He attended Fort William Collegiate Institute and the University of Toronto. During World War II he was a medical officer in the R.C.A.F., and after the war practised in Port Arthur for a short time before he moved to Toronto where he established practice as a specialist in anæsthetics.

Dr. White is the son of the late Dr. J. H. White, former medical officer of health for Fort William. He is survived by his widow and two children.

FORTHCOMING MEETINGS

CANADA

CANADIAN ASSOCIATION OF OCCUPATIONAL THERAPY, 25th Annual Convention, Royal York Hotel, Toronto, Ont. (Miss Joy Miles, C.A.O.T., 331 Bloor Street West, Toronto 5.) October 29-31, 1955.

MONTREAL MEDICO-CHIRURGICAL SOCIETY, 23rd Annual Fall Clinical Convention, Montreal, Quebec. (Mrs. Gerda E. Copp, Executive Secretary, Suite 718, 1538 Sherbrooke St. West, Montreal 25, Que.) October 31-November 5, 1955.

CANADIAN PUBLIC HEALTH ASSOCIATION, Laboratory Section, Annual Christmas Meeting, Royal York Hotel, Toronto, Ontario. (Dr. F. O. Wishart, Secretary, Laboratory Section, C.P.H.A., 150 College St., Toronto 5, Ont.) December 12-13, 1955.

SOCIETY OF OBSTETRICIANS AND GYNÆCOLOGISTS OF CANADA—1956 Annual Meeting, Manoir Richelieu, Murray Bay, Quebec. (Dr. F. P. McInnis, Secretary, Society of Obstetricians and Gynæcologists of Canada, 1230 Avenue Road, Toronto, Ont.) June 8-10, 1956.

CANADIAN MEDICAL ASSOCIATION, 89th Annual Meeting, Ecole de Commerce, Quebec, Quebec. (Dr. A. D. Kelly, General Secretary, Canadian Medical Association, 244 St. George Street, Toronto 5, Ont.) June 11-15, 1956.

UNITED STATES

AMERICAN HEART ASSOCIATION, Annual Meeting and Twenty-Eighth Annual Scientific Session, Jung Hotel, New Orleans, Louisiana. (The Medical Director, American Heart Association, 44 East 23rd Street, New York 10, N.Y.) October 22-26, 1955.

INTER-SOCIETY CYTOLOGY COUNCIL, 3rd Annual Meeting, Statler Hotel, Cleveland, Ohio. (Dr. P. F. Fletcher, Secretary-Treasurer, 634 N. Grand Blvd., St. Louis 3, Mo.) November 11-12, 1955.

AMERICAN PUBLIC HEALTH ASSOCIATION, INC., 83rd Annual Meeting and Meetings of Related Organizations, Kansas City, Missouri. (The American Public Health Association, Inc., 1790 Broadway, New York 19, N.Y.) November 14-18, 1955.

NATIONAL SOCIETY FOR CRIPPLED CHILDREN AND ADULTS, Annual Convention, Palmer House, Chicago. (Director of Information, 11 South LaSalle Street, Chicago 3, Illinois.) November 28-30, 1955.

NEW YORK HEART ASSOCIATION, Conference on Rheumatic Fever and Heart Disease, Biltmore Hotel, New York. (New York Heart Association, 485 Fifth Avenue, New York 17, N.Y.) November 29, 1955.

AMERICAN MEDICAL ASSOCIATION, Clinical Meeting, Boston, Massachusetts. (Dr. George F. Lull, 535 North Dearborn Street, Chicago 10, Illinois.) November 29-December 2, 1955.

AMERICAN PSYCHOSOMATIC SOCIETY, 13th Annual Meeting, Sheraton Plaza Hotel, Boston. (Dr. S. Cobb, Chairman, Programme Committee, 551 Madison Avenue, New York 22, N.Y.) March 24-25, 1956.

INTERNATIONAL ANÆSTHESIA RESEARCH SOCIETY CONGRESS, Flamingo Hotel, Miami Beach, Florida. (Dr. T. H. Seldon, Mayo Clinic, Section on Anæsthesiology, Rochester, Minn.) April 9-12, 1956.

WORLD CONFEDERATION FOR PHYSICAL THERAPY, Second International Congress, New York. (Miss M. J. Neilson, Secretary-General, c/o Chartered Society of Physiotherapy, Tavistock House South, Tavistock Square, London, W.C.1, England.) June 17-23, 1956.

OTHER COUNTRIES

INTERNATIONAL ACADEMY OF LEGAL MEDICINE AND SOCIAL MEDICINE, Fourth International Congress, Genoa, Italy. (Professor P. Dervillée, 159 rue de la Croix de Seguey, Bordeaux, France.) October 13-17, 1955.

NUTRITION SOCIETY—Symposium, London, England. (Dr. R. J. L. Allen, Monkhouse and Glasscock Ltd., London, S.E.1, England.) October 15, 1955.

CONGRESS OF THE INTERNATIONAL UNION OF THE MEDICAL PRESS, Paris, France. (Jean Mignon, Secretary-General, "Le Concours Médical," 37 rue de Bellefond, Paris 9e.) October 16-20, 1955.

MEDICAL ASSOCIATION OF SOUTH AFRICA—40th Medical Congress, Pretoria, South Africa. (Joint Organizing Secretaries, Room 28, Administrative Building, General Hospital, Pretoria, South Africa.) October 17-22, 1955.

PAN-AMERICAN CONGRESS, International Congress of Surgeons (in conjunction with Argentine conference on thoracic surgery), Mendoza, Argentina. (Biblioteca, Asociacion Medica Argentina, Santa Fé 1171, Buenos Aires, Argentina.) October 22-26, 1955.

ASSOCIATION OF ANÆSTHETISTS OF GREAT BRITAIN AND IRELAND—Annual Meeting, London, England. (Association of Anæsthetists of Great Britain and Ireland, 45 Lincoln's Inn Fields, London, W.C.2, England.) November 3, 1955.

PHYSIOLOGICAL SOCIETY — Meeting, London, England. (Professor A. A. Harper, Dept. of Physiology, King's College, Newcastle-upon-Tyne, England.) November 4-5, 1955.

INTERNATIONAL CONGRESS OF ALLERGOLOGY, Rio de Janeiro, (Dr. F. W. Wittich, 424 LaSalle Medical Bldg., Minneapolis, Minn.) November 6-12, 1955.

SOCIETY OF THORACIC SURGEONS OF GREAT BRITAIN AND IRELAND, Glasgow, Scotland. (Mr. J. Leigh Collis, F.R.C.S., 15 Highfield Road, Edgbaston, Birmingham, England.) November 11-12, 1955.

SIXTH VENEZUELAN CONGRESS OF MEDICAL SCIENCES, Caracas, Venezuela. (Dr. A. L. Briceno Rossi, Apartado 4412, Ofic. del Este, Caracas, Venezuela.) November 18-26, 1955.

BRITISH ASSOCIATION OF SPORT AND MEDICINE—Meeting, St. Thomas's Hospital, S.E.1, London, England. (Dr. D. J. Cussen, British Association of Sport and Medicine, 95 Mount Street, London, W.1, England.) November 21, 1955.

BRITISH ASSOCIATION OF PLASTIC SURGEONS, Annual Meeting, London, England. (British Association of Plastic Surgeons, 45 Lincoln's Inn Fields, London, W.C.2, England.) December 9-10, 1955.

PHYSIOLOGICAL SOCIETY—Meeting, London, England. (Professor A. A. Harper, Department of Physiology, King's College, Newcastle-upon-Tyne, 1, England.) December 16-17, 1955.

INTERNATIONAL CONGRESS FOR THE SOCIAL REHABILITATION OF THE LEPER, Rome, Italy. (M. F. Sarsale, International Congress for the Rehabilitation of the Leper, Via Condotti, Palazzo Malta, Rome, Italy.) April 16-18, 1956.

INTERNATIONAL UNION FOR PUBLIC HEALTH EDUCATION, Third Conference, Rome, Italy. (Mr. Lucien Viborel, Secretary-General, 92 rue St. Denis, Paris Ier, France.) April 27-May 5, 1956.

INTERNATIONAL FERTILITY ASSOCIATION, SECOND WORLD CONGRESS, Naples, Italy. (Prof. G. Tesauro, President of Committee Arrangements, S. Andrea delle Dame, 19, Naples.) May 1956.

NINTH WORLD HEALTH ASSEMBLY, Geneva, Switzerland. (World Health Organization, Palais des Nations, Geneva, Switzerland.) May 9, 1956.

INTERNATIONAL MEDICO-ATHLETIC FEDERATION, 11th Congress, Buergenstock, Switzerland. (Dr. G. Schoenholzer, Secretary-General, Bluemlisalpstr. 7, Muri-Berne, Switzerland.) May 29-June 1, 1956.

NEWS ITEMS

MANITOBA

Dr. Maxwell Bowman, provincial director of preventive medical services, has been appointed a fellow of the newly established American College of Preventive Medicine. This fellowship, the first to be awarded in this province, was given in recognition of his work.

At the International Northern Great Plains Conference on rehabilitation at the University of Manitoba, Dr. Maynard C. Richards urged a comprehensive service for mentally retarded children. Dr. Richards is director of the psychoeducational clinic at the University of Minnesota. He believes that public schools for mental deficients can be and should be maintained. For adult mentally retarded persons sheltered workshops are helpful.

Dr. G. H. Hamblin has been elected president of the newly formed Portage la Prairie Lions Co-operative recreational centre.

The Federal Health Minister has announced that a grant of $114,820 will be made to assist in the construction of a new hospital at Portage la Prairie. It is scheduled for completion in June 1956, and will accommodate 90 patients and laboratory and x-ray departments. The old Portage General Hospital has been condemned for further use because of age and deterioration.

Ross MITCHELL

NEW BRUNSWICK

Dr. F. A. McGrand of Fredericton Junction has been appointed to a seat in the Senate of Canada. Dr. McGrand has served his province as a physician, County Councillor, Member of the Provincial Legislature, Speaker of the House and Minister of Health.

After three years' postgraduate study in surgery in Montreal and London, England, Dr. G. F. W. Moore has resumed his practice in Hartland, N.B.

Dr. J. A. Melanson, Chief Medical Officer of the New Brunswick Department of Health, has been elected a Fellow of the American College of Preventive Medicine.

Dr. A. M. Clarke and Dr. G. E. Maddison attended a meeting of the Health Planning Committee for the Maritimes and Newfoundland in July at St. John's, Newfoundland, as representatives of the New Brunswick Department of Health.

Dr. A. F. Chaisson, Director of Communicable Disease Control for New Brunswick, reports that 25,000 children in grades one and two have received their first two doses of Salk vaccine.

The contract for the construction of a new building for Saint Joseph's Hospital at Saint John, N.B., was awarded to Anglin-Norcross Maritimes of Montreal on August 12 for a total amount of $2,363,883. This building will be completed in 1957 and represents a great addition to the medical centre in Saint John.

Dr. R. C. Eaton has resigned his position as Superintendent of the Provincial Hospital at Campbellton.

Dr. A. K. Carton of St. Stephen has received the Diploma of the American Board of General Surgery.

Dr. Walter J. Fisher of Saint John attended the meeting of the Canadian Neurological Society in Toronto.

A. S. KIRKLAND

QUEBEC

Dr. Roger Dufresne has been appointed assistant dean of the faculty of medicine, University of Montreal. Dr. Dufresne, who succeeds Dr. Roméo Pepin, is a graduate of the university and has done postgraduate work at Paris and Boston universities. He has been on the university staff since 1945.

Other new medical faculty appointments and promotions are as follows: Dr. Paul Robert to the rank of professor; Drs. Sorin Sonea, Marcel Lamoureux, Louis Poirier, Bernard Baillargeon, Rosario Robillard, Paul David, Jean Grignon, Gustave Gingras and Roland Cloutier, all to the rank of associate professor. Also appointed to the rank of assistant professor were Drs. Jean Marie Bourgault, Luc Vaillancourt, Jacques Genest, Jean Gratton, Roger Demers, Jacques Gelinas, Jean Marie Lessard, Gerard Mignault, Martial Bachaud, René Charbonneau, Edouard Laberge and Lucien Panaccio.

Dr. C. D. Shortt, senior medical officer of the Canadian National Railways, has been elected president of the Industrial Medical Association of the Province of Quebec.

A report from our Department of Health shows that the struggle against tuberculosis is winning all along the line. Treatment facilities have never been more adequate or effective. The most remarkable improvements have been in infections in other organs than the lungs. Until streptomycin was added to the list of anti-tuberculosis drugs, tuberculous meningitis used to be 100% fatal. Now about 60% of patients survive and some recover completely. The death rate in bone and joint tuberculosis has dropped to less than 20%, and mortality from genito-urinary tuberculosis has been halved.

New drugs have played a prominent role not only in combating the bacterial infection, but also in making better surgery possible under the antiseptic cover conferred by the antibiotics. The most prominent of these antibiotics are streptomycin, *p*-aminosalicylic acid (PAS) and isoniazid. The tubercle bacilli have shown that they can soon develop resistance to these agents, but new combinations of the drugs seem to be more effective and have fewer side-effects.

The Canadian Mental Health Association, at its last annual meeting in Montreal, elected Mr. J. S. D. Tory, O.B.E., Q.C., Toronto, president, succeeding Dr. Jonathan C. Meakins, C.B.E. A presentation was made to Dr. Meakins, who had served since 1946. Tribute was also paid to Dr. Margery King of Toronto, who was executive officer of the Fifth International Mental Health Congress in Toronto last year.

A. H. NEUFELD

Dr. Alan B. Noble has been appointed anæsthetist-in-chief at the Royal Victoria Hospital, Montreal, succeeding Dr. F. A. H. Wilkinson who has resigned that post because of ill health. Dr. Noble is a native of Ontario and a graduate of the University of Toronto. After interning at the Toronto General Hospital he engaged in private practice in Brampton, Ont., for five years before taking his training in anæsthesia in various Montreal hospitals. Since 1946 Dr. Noble has been the chief anæsthetist at the Hôtel-Dieu Hospital, Kingston, and lecturer at Queen's University. He is President of the Canadian Anæsthetists' Society. Dr. Noble will take up his duties on November 1.

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ARMED FORCES

A review of the assisted university training plans for medical students in the Royal Canadian Navy reveals that 22 medical officers of the Regular Force have been enrolled through this source over the past five years. These medical officers are now serving in the fleet and fleet establishments, or in some cases have returned to training schools for planned postgraduate training. Five additional medical officers have commenced required internship training this year and will be available for duty next summer. Four students will commence their final year of medicine this fall, while additional students have been enrolled in the present fiscal year and will be available for duty in 1957.

The results of the assisted training programme have been most gratifying and necessary inasmuch as the plan has produced a steady flow of young medical officers. Medical officers derived from this source now represent over 50% of the total number of the Regular Force of the R.C.N.

Colonel E. E. Tieman, O.B.E., O.D., M.D., formerly Command Medical Officer, Eastern Command, Halifax, has begun training at the University of Toronto leading to the Diploma in Public Health.

Major J. T. Baird, M.B., Ch.B., D.O.M.S., is taking further training in ophthalmology at Sunnybrook Hospital, D.V.A., Toronto.

Major D. E. Yates, M.B., B.S., is on an assistant residency in orthopædic surgery at Deer Lodge Hospital, D.V.A., Winnipeg, Man.

Captain A. R. S. Cumming, M.D., commenced training in psychiatry at Queen Mary Veterans' Hospital, Montreal, on July 1, 1955.

Captain S. M. Mazewski, M.B., Ch.B., B.A.O., is undertaking training in otolaryngology in Toronto hospitals.

Captain W. R. Woodley, M.D., began training in radiology July 1, in Shaughnessy Hospital, D.V.A., Vancouver, B.C.

Captain W. J. Vail was promoted to the rank of Major effective July 1, 1955.

Twenty-two medical students enrolled as undergraduates with the rank of Second Lieutenant have obtained their M.D. degree and are now employed in the R.C.A.M.C. They will be promoted to the rank of Captain when they receive their licences to practise.

Wing Commander H. J. Bright, former Staff Officer Medical Services, Training Command, Trenton, Ontario, has been transferred to No. 1 Air Division Headquarters, France, as Staff Officer Medical Services, replacing Wing Commander W. J. Taylor who has begun a two-year postgraduate course in radiology at the University of Toronto.

Wing Commander Ian Barclay has been transferred from No. 5 Air Division Headquarters, Vancouver, to Training Command Headquarters, Trenton, where he has assumed the duties of Staff Officer Medical Services. Squadron Leader G. B. Bending has replaced Wing Commander Barclay as Staff Officer Medical Services, No. 5 Air Division Headquarters, Vancouver.

BOOK REVIEWS

PATHOLOGY FOR THE SURGEON

W. Boyd, Lecturer on the Humanities in Medicine, The University of Toronto. 7th ed. 737 pp. Illust. W. B. Saunders Company, Philadelphia and London, 1955.

"Better than ever" must be the verdict on this new and greatly altered edition of what used to be called Boyd's *Surgical Pathology*. The author explains that "an old suit may be patched and repaired so as to make it reasonably presentable, but the time comes when it begins to get dated." He has therefore rewritten the volume, having mainly in mind the graduate, the surgeon, and the younger man, in particular the intern or resident needing to refresh his memory of pathology for specialty board examinations. Many older men will no doubt also want to profit by the agreeably imparted wisdom in these pages.

The author explains why he has changed the title, to correspond with the changing orientation of the surgeon to pathology; he has introduced new material on the relation of symptoms to lesions, on the lines of his *Pathology of Internal Diseases.*

In line with the broadening in scope of the surgical and pathological field, new chapters appear on the cardiovascular system, the endocrines, the skin, the soft tissues and wound infections. The chapter on the general pathology of tumours has undergone much change, and is a notable exposition of the subject.

The references have been brought up to date, and useful synopses of chapters have been added under each chapter heading.

The brilliance of expression is undiminished. Dr. Boyd speaks of erysipelas spreading "as a drop of grease spreads on a piece of paper", and of the centre of the lesion "as if a wind had spread over a cornfield". In his tribute to Maude Abbott he says that she was "consumed by a fire kindled by a spark from Osler". Of the vexed question of carcinogenesis "the trouble is that we probably swim in a sea of carcinogens". He likens the histological picture in the involuting breast to a Beethoven sonata with a main theme and variations.

The surgeon who fails to make acquaintance with this book will be very much the poorer for his omission. Reading it is an intellectual treat.

BLUTGERINNUNGSFAKTOREN
(Factors in Blood Coagulation.)

E. Deutsch, Instructor at the Clinic for Internal Medicine of the University of Vienna. 298 pp. Illust. 42 Marks. Franz Deuticke, Vienna, 1955.

This monograph is the first volume of a new series of "Monographs of Biochemistry" edited by Hoffman-Ostenhof in Vienna. As the author points out in the foreword, most of the progress in our knowledge about blood coagulation during the last 20 years is due to the results of biochemical research methods. The present volume deals with this aspect of the problem, while the clinical side has been discussed by the author elsewhere. The text is clearly written and contains numerous descriptions of methods for the demonstration of various clotting factors. There is also a discussion of other enzymes with an action on blood clotting, as for example, trypsin, snake poisons and others.

The world literature up to June 1954 has been considered and over 1,600 references are cited. The nomenclature follows in general that of Owren, but synonyms are mentioned throughout the text and are collected in the form of a table at the end of the volume.

The book will be of value to all German-reading physicians and laboratory workers interested in this problem.

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THE SPINE

A Radiological Text and Atlas.

B. S. Epstein, Chief, Department of Radiology, The Long Island Jewish Hospital, New Hyde Park, New York. 539 pp. Illust. $16.50. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1955.

This first edition of a treatise on the spine is well written, pleasant to read and amply illustrated. The author has undertaken an extensive collection of the literature, reviewed it critically and formulated a bibliography that is one of the most desirable sections of the whole volume.

The opening chapter deals with the normal spine. The embryology is well described, but figures to clarify the text are lacking. In such a detailed, specialized subject, where so many of the congenital abnormalities can be understood on a basis of deviation of the normal developmental pathway, a clear grasp of the embryology is highly desirable. This is difficult to obtain if one is to use this volume as a "text". In contradistinction to this, the section on the architecture of the spinal column is well written and illustrated. Several important clinical points are noted, including the motion of the cervical articular facets in flexion and extension.

Though discography is still a subject of much discussion, several figures of normal and pathological discograms would have been in order.

The various malconfigurations of the spinal column, as well as the diseases of congenital, endocrine and metabolic origin, are well presented in chapters two and three, with good figures to illustrate the text. One wonders whether such non-specific disease entities as arachnodactyly merit mention in such a volume. The section on scoliosis is grossly inadequate and needs great revision. Most glaring of the omissions is absence of discussion on the criteria of measurement of the scoliotic curve.

Chapter four deals with the inflammatory, degenerative and toxic diseases of the spine. The section dealing with tuberculosis does not emphasize several points. Marginal rarefaction of the vertebral body, as the very earliest evidence of the disease, has not been given its just importance. The enormous problem encountered in the radiological interpretation of the treated patient's radiographs is not mentioned at all. Criteria of healing are not given sufficient emphasis; to be sure, bony ridges are secure evidences of repair, but these are not always present. The young radiologist should not leave this section feeling that myelography of a suspect tuberculous spine is desirable and without immense danger.

The discussion of chordomas in chapter five is complete and to be recommended. The incidence and local problems of the tumour are presented in a very clear and concise manner. The section on traumatic changes is well written, abundantly illustrated and complete in its scope. The material on spondylolisthesis is of the highest quality.

The remaining chapters on diseases of the intervertebral discs, of the spinal cord and its coverings, and of the hæmatopoietic, collagen and reticulo-endothelial systems deal adequately with the subjects. The last chapter on aortic aneurysms allows for completion of the discussion on diseases of the spine.

The bibliography is one of the most important aspects of the book. It is organized alphabetically and complete in detail; the author has done a great service to the reader in collecting so much literature. It is a very good source of reference to original articles.

In spite of the criticism offered above, this volume is a desirable text on diseases of the spine. The author has written in clear fashion and used abundant illustrations. It is to be recommended to all people who deal with disorders of the spinal column.

PATHOLOGY

P. A. Herbut, Professor of Pathology, Jefferson Medical College, and Director of Clinical Laboratories, Jefferson Medical College Hospital, Philadelphia, Pennsylvania. 1,227 pp. Illust. $16.00. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1955.

This new textbook of pathology is a book which tends to grow on the reader. At first, Dr. Herbut's very terse style and his grim determination to include everything about disease in one volume make for a little difficulty in reading, but once the reader has got used to this he will begin to appreciate the immense industry that has gone into the compilation, and the thought that must have gone into the compression of material. Dr. Herbut says that his book is intended for the student of medicine —undergraduate or postgraduate, and that in order to reduce an encyclopædic amount of information into the confines of a single volume, a rather rigid pattern has had to be followed and the material presented without adornment. He has undoubtedly produced a very useful text, in which not only the morbid anatomy and physiology of disease conditions are described but a thumbnail sketch of the clinical features and sometimes the treatment and prognosis are included.

The arrangement of material follows fairly conventional lines, the chapters on special pathology being subdivided into the basic pathological processes of congenital anomalies, degenerations, inflammations, physical disturbances and tumours. There is a valuable addition to the customary text in the form of a chapter on the autopsy, which also contains a handy list of weights and measurements of normal adult organs. The illustrations deserve special praise. There are enormous numbers of them, and they have not been reduced inordinately but kept to a convenient size for study of histological detail. Dr. Herbut has also made an effort to enlighten the student as regards etymology of terms used in pathology. The author provides the reader with a good bibliography for further study.

This very concise text certainly represents value for money in terms of the amount of information conveyed. In addition to its employment by students, it would be very handy for rapid reference, since it contains a remarkable amount of detail in a small compass, and appears to omit nothing in the field of medicine.

LISTERIOSIS

Dr. Heinz Seeliger, Hygiene Institute of the Frederick-Wilhelm University, Bonn, West Germany. 152 pp. Illust. 11.60 Marks. Johann Ambrosius Barth, Leipzig, 1955.

Recent papers in the *Canadian Medical Association Journal* have shown that human listeriosis, or infection with *Listeria monocytogenes*, is probably much more prevalent in Canada than the average physician thinks, and is often undiagnosed. In Germany, indications are that the infection is also widespread and misdiagnosed, as indeed it probably is all over the world. The present monograph from Bonn gives a remarkably complete review of what is known of the etiological agent and the infection in both animals and man. Particular emphasis is laid on the technique of diagnosis, which can only be made bacteriologically or—to a lesser extent—serologically. The author has assembled a very full and up-to-date bibliography, and the work merits study by bacteriologists and clinicians everywhere. It is pleasing to see a portrait of Professor E. G. D. Murray of Montreal included. He has done much to promote interest in this comparatively obscure but important subject.

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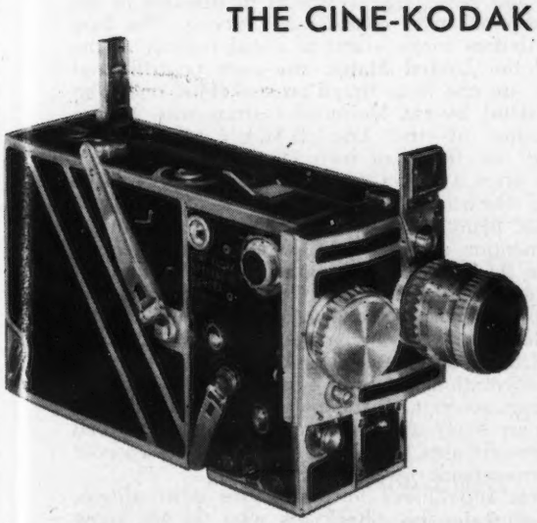

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SURGICAL FORUM

Proceedings of the Forum Sessions, Fortieth Clinical Congress of the American College of Surgeons, Atlantic City, N.J., November 1954. Chairman, H. B. Shumacker, Jr., Indianapolis. 851 pp. Illust. $10.00. W. B. Saunders Company, Philadelphia and London, 1955.

This volume contains the papers presented at the Clinical Congress of the American College of Surgeons in November 1954. The papers are grouped under the following headings: heart and great vessels; blood vessels and circulation; vascular grafts; œsophagus, stomach and intestines; liver and pancreas, nutrition, body fluids and metabolism; steroids and cancer; anæsthesiology; burns; shock and wounds; and plastic anatomic casts.

The papers are all quite short and concise, with a summary, and with appropriate bibliography. A high percentage of the papers consists of reports of experimental work being done in various North American surgical centres. The experimental work is directly related to clinical problems. The remaining papers are direct reports of clinical work.

One criticism of the volume might be that more careful editing of some of the papers would have eliminated certain superfluous language and grammatical errors, and thus would have made these papers more easily and clearly understood. For example, one paper ends as follows: "It is concluded that while a diminished adrenocortical response may be observed not uncommonly in chronically ill subjects, it must be rare that these subjects cannot survive surgery without replacement therapy."

As Dr. I. S. Ravdin states in the foreword: "The papers presented in this volume will give those who read them an understanding of many of the biochemical and physiological problems which now concern the surgical subject." The volume should appeal to those interested in recent advances in surgery.

SEGMENTAL ANATOMY OF THE LUNGS

A Study of the Patterns of the Segmental Bronchi and Related Vessels.

E. A. Boyden, Professor of Anatomy (Emeritus), the Medical School, University of Minnesota. $15.00. The Blakiston Division, McGraw-Hill Book Company, Inc., New York, Toronto, London, 1955.

Segments are lobar subdivisions of irregular size and shape which may be resected to remove a lesion, and the importance of their anatomy has grown with chest surgery. Each is a somewhat pyramidal block with pleural base and hilar apex. The lateral "boundaries" or "intersegmental planes" are artificial, and their operative fission relies on relative freedom from intercrossing vessels. To make this book, Boyden and his numerous associates at the University of Minneapolis worked nine years in dissecting hundreds of segments in specially prepared lungs. They have made rich use of roentgenography, bronchoscopy and the experience of surgeons. The beginner might start with development, for in human embryos of 13 mm. all 10 segments of each lung are indicated on bronchial tree models as end buds of the segmental bronchi, and on the lung surface most appear as elevations. He might then read Chapter 10—a unique atlas of 11 magnificent coloured plates revealing the gross structure of bronchi and associated blood vessels, to provide an approach to the segments from the hilum. Sometimes the view is with the lobes spread apart at a fissure. In Chapter two and elsewhere the pleural aspects are graphically presented and interpreted in reference to the bronchial tree. The nomenclature of Jackson and Huber predominates. The segments are individually described and illustrated in Chapters three to eight, each around the "axis" of its shrublike segmental bronchial system with pulmonary arterial and venous systems integrated. Prevailing patterns and variations are given and surgical hints abound. Tables are numerous, illustration and documentation profuse. Simple letter and number designations denoting correlated bronchi, arteries and veins in a segment are used, as B^1, A^1, V^1 respectively. An historical review is provided and the work of the Brompton Hospital in the evolution of segmentology stressed. There is much quantitation. This book is an essential for the thoracic surgeon.

PRACTICAL MEDICAL MYCOLOGY

E. L. Keeney, formerly in charge of the Mycology Laboratory, Visiting Physician and Dispensary Physician (Allergy), The Johns Hopkins Hospital, Baltimore, Maryland. 145 pp. Illust. $5.00. Charles C Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1955.

This book presents the whole field of medical mycology. Each disease is dealt with by giving the historical background, geographical distribution, incidence, sources of infection, symptomatology, mycology, prognosis and treatment. A one-page diagrammatic illustration recapitulates the main features of each disease and its causative organism. There also are chapters on fungi as allergens and fungus poisoning.

The book is written in a simple style and is easy to read. The author has tried to omit as many technical terms as possible; in some respects, this simplification has gone too far, therefore the book cannot replace any of the more elaborate existing textbooks, nor can it be used for the laboratory work always necessary to establish a correct diagnosis of a fungus infection. However, for a practising physician, the book presents a good introduction to medical mycology. This subject unfortunately appears to those with little training in the field to be rather complicated because the use of many inexact, misleading terms and too great a number of synonyms for the pathogenic fungi.

A TEXTBOOK OF MEDICINE

Edited by R. L. Cecil, Professor of Clinical Medicine, Emeritus, Cornell University, New York, and R. F. Loeb, Bard Professor of Medicine, Columbia University, New York. 9th ed. 1,786 pp. Illust. $15.00. W. B. Saunders Company, Philadelphia and London, 1955.

In the ninth edition of this well-known textbook, the authors have continued their policy of incorporating the biochemical and physiological aspects of diseases in the clinical descriptions with considerable success. The long list of contributors forms a sort of social register of the internists of the United States; the only contributions from outside are one from Brazil on snakebite, one from London, England, by Dr. Desmond Curran, who was on the programme of the late B.M.A.-C.M.A.-O.M.A. meeting, and one from our own Dr. Wallace Graham. Dr. Graham gives a sound summing up of the fibrositis syndrome on which he is an authority.

Among the many new contributions to this edition, we might mention Dr. Chester Keefer's new general discussion on the bacteriology, pathology and immunology of bacterial disease, a good section on Coxsackie virus diseases, a rearrangement and expansion of the section on leptospiroses, new material by Dr. Spies on vitamins and blood regeneration, and kwashiorkor, a discussion of dehydration and fluid balance by Dr. Berliner, and a succinct account of the painful shoulder by Dr. Cecil. There are many other new sections, some of which deal with new diseases such as a cat-scratch disease or epidemic hæmorrhagic fever.

It is almost superfluous to praise this new edition, since there must be few physicians who do not know of the book. If there are any who are still unacquainted with it, we would suggest to them that it would be well worth their while to look over this beautifully presented text.

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LECTURES ON THE SCIENTIFIC BASIS OF MEDICINE. Vol. III, 1953-54.

British Postgraduate Medical Federation, University of London, England. 398 pp. Illust. 35/-. The Athlone Press, University of London, England, 1955.

This is the third volume in which is reprinted a series of lectures given by the British Postgraduate Medical Federation of the University of London. The purpose of the lectures is to help graduates to keep abreast of the broad trends in scientific fields other than their own, and thus to provide a valuable antidote to narrowmindedness. In the present volume, perhaps the most entertaining item is J. B. S. Haldane's lecture on the genetics of some biochemical abnormalities, although his discussion on sickling may need modification in the light of more recent research.

Clinicians will be interested in Professor Garrod's lecture on the causes of failure in antibiotic therapy, a theme which he also expounded at the recent Toronto meeting. They will also enjoy Dr. Mackenna's contribution on the scientific approach to dermatology. Most of the other lectures deal with more difficult themes in experimental pathology or pharmacology, such as tissue repair, biological synthesis, anticholinesterases, acetylcholine and cardiac rhythm, growth hormone, sex hormones, phosphatases in disease, body water control, and chemotherapy of cancer. There is a useful chapter on antiviral immunity, and an interesting one on hemispherectomy and localization of function.

GYNAEKOLOGISCHE AND ROENTGEN-DIAGNOSTIK (Gynæcological Diagnostic Radiology)

G. K. F. Schultze and J. Erbslöh. 399 pp. Illust. 2nd ed. Ferdinand Enke, Stuttgart, 1954.

This German text gives an excellent detailed outline of the technique, indications, pitfalls, and benefits of modern hysterosalpingography. This is a diagnostic technique widely used in the study of sterility but not often enough in puzzling pelvic conditions. More liberal use of the methods outlined herein, in problems where physical examination and diagnostic curettage are negative, will lift a high percentage of such cases out of the "functional" or "endocrine" groups.

There are step-by-step descriptions of the equipment and procedures used, with excellent illustrations. Over 200 x-ray plates are included in a very clear-cut manner and they alone could easily function as an illustrative atlas on the subject, even to readers with only a smattering of German. The variations obtained with change of position and with the newer water-soluble materials are compared.

A 47-page bibliography of the world literature on the subject to 1953 is included. There is, however, no reference to Decker's very useful intrauterine rubber catheter which the reviewer has used extensively after negative curettage and which is very useful where patients have to be transported out of the operating room for radiological study elsewhere.

An English translation would find a wide audience.

PRACTICAS DE FISIOLOGIA

E. Briese, Profesor Agregado. 232 pp. Illust. Universidad de Los Andes, Instituto de Fisiologia, Mérida, Venezuela, 1955. Publicacion No. 43.

This is an elementary text in practical physiology published by the University of the Andes, Mérida, Venezuela. It includes all the usual experiments on frog, mammal and man, well described and illustrated, and carefully chosen so as to interest the student and make him think.

MODERN TRENDS IN OPHTHALMOLOGY

Third Series. Edited by A. Sorsby. 346 pp. Illust. $13.00. Butterworth & Co. Ltd., London; Butterworth & Co. (Canada) Ltd., Toronto, 1955.

The first two volumes of "Modern Trends in Ophthalmology" have been useful contributions to the ophthalmic literature. This, the third volume, is slightly smaller than the previous two, but it maintains the original high standards. Short articles by various authors cover many fields.

The application of phase-contrast miscroscopy to the eye is described by J. François and M. Rabaey, electron miscroscopy by J. François, M. Rabaey, and G. Vandermeerssche, and histochemical techniques by J. L. Reis. These chapters form an excellent summary of this experimental work. In this first section of the book E. H. Bárány presents a mathematical discussion of the measurement of an aqueous flow in the experimental animal.

Under the heading of Diagnostic Procedures there is an excellent discussion by Milton L. Berliner on the slit-lamp miscroscopy of the posterior segment of the eye. This is beautifully illustrated. It adequately describes the methods of examination and the common pathological findings in the vitreous and in the fundus. In the same section the new technique of tonography is described by W. Morton Grant, ophthalmoscopy of the extreme fundus periphery by C. L. Schepens, and orbitonometry by E. C. Copper.

One of the interesting features of the book is the prominence given to hereditary affections of the eye. Diagnostic criteria of genetic affections are discussed by Harold F. Falls, who also considers intermediate sex-linkage and its clinical applications; the nature of malformations, congenital cystic sex-linked retinal detachment, and generalized fundus dystrophy are described by Arnold Sorsby.

In the field of experimental pathology there are excellent discussions of the current aspects of ocular pharmacology by Irving H. Leopold and of the action of x-rays and radioactive substances on ocular tissues by David G. Cogan. An up-to-date review of pharmacology should be an important feature of any book on modern trends. Leopold has reviewed this subject in a short but satisfactory manner.

There is a comprehensive review of muco-cutaneous ocular syndromes by Phillips Thygeson. This, combined with discussions of ocular aspects of diffuse collagen disease by Manuel L. Stillerman, of allergy by Irving H. Leopold and Howard G. Leopold, and of cortisone and ACTH in diseases of the eye by Arnold Sorsby and Mary Savory, forms a very valuable part of the book.

Under a section entitled "Treatment", Sorsby discusses the newer antibiotics. Corneal transplantation is considered by H. P. Stallard and lamellar sclerectomy for rentinal detachment by C. Dee Shapland. Keith Lyle and A. G. Cross discuss abnormalities of vertically acting extrinsic ocular muscles, and W. H. Fink describes the surgery of the oblique muscles. A number of short articles present some recent advances in surgical technique.

At the end of the book is a valuable section on social aspects of ophthalmology. Arnold Sorsby and Victoria A. Spenser discuss blindness in England and Wales. Problems of the visually handicapped are considered by T. H. Carruthers and P. McGregor Moffatt.

This book is well produced, in clear type and high gloss paper. It has an adequate index. The art work and production of the illustrations is good. Because of the many authors there is some variation in the quality of the different sections, but the material is good, well chosen, and well presented.

THE DISEASES OF OCCUPATIONS

D. Hunter, Physician, the London Hospital. 1st ed. 1,007 pp. Illust. Five guineas. English Universities Press Limited, 102 Newgate St., London, E.C. 1, 1955.

The purpose of this publication, as given in the preface, is to review with emphasis on clinical aspects the problem of disease in relation to occupation, and to contribute to the establishment of the subject on an academic basis. This has been accomplished, and in such a way that it should be a stimulus to more widespread medical interest in the subject. The book reflects the author's reputation as an internist, clinical teacher and research worker with wide interests and a gift for expressing himself. It is replete with short biographical notes, suggestive sub-headings and analogies; for example, the conditions of polymerization of ethylene temperatures around 200° C. and pressures varying between 1,000 and 1,500 atmospheres were similar to those produced in a 15-inch naval gun.

As the title suggests, the material is drawn from experience including not only manufacturing industry but also agriculture, fisheries, the armed services and the too often neglected laboratory services. The development of the contribution by physicians and others to the knowledge of these diseases and the increasing contact with industry of the general practitioner are described, including the details of the programme for the maintenance of general health which came with his part-time or full-time appointment to industry. It is desirable that the general health of the worker should be discussed in a publication on diseases of occupation, since medical control of these diseases is inseparable from it.

The author outlines the awakening to the importance of experimental science, particularly chemistry and physics, and the application of the results to the production of goods and services, with its political and social implications; the gradual changes in public opinion toward abuses in employment and the struggle for legislation to correct them. "The industrial revolution saw the birth of organic chemistry."

Steps in the growth of industrial processes, and the early observations on diseases arising out of occupation, are extended to descriptions of modern processes sufficient to explain the reason for the medical findings in those exposed.

The occupational diseases for consideration are selected by cause from groups such as metals, organic solvents, mineral or vegetable dusts, covering those which have been or are most likely to be encountered. Fortunately the number of classical cases of most of these diseases is not high in relation to that for non-occupational diseases. In the nature of the subject the opportunities for observation are apt to be sporadic and their significance easily missed. Where control measures are inadequate for any reason, the general practitioner or the industrial physician is the first line of defence, so that the author emphasizes the early effects of exposure—what to look for, knowing the exposure, or, at least as important, what may arouse suspicion that occupation is a factor. Exception might be taken to the statement that "in practice carbon tetrachloride is one of the least harmful of the chlorinated hydrocarbons" but the author does attempt to determine in detail what exposure occurred to produce the cases observed and what effect, if any, was produced on fellow workmen. Where possible, common factors are discussed, for example, the substances which produce methæmoglobin, or the chlorinated aliphatic hydrocarbons which cause liver damage. A feature of this work is the welding together of the physician's general activities in industry, occupational and medical history, appraisal of exposures, incidence of disease, pathology, signs and symptoms with clinical evaluation, relevant research, medical and engineering control and treatment, in almost story-book fashion.

The author's selection of reports from the literature and his personal experience have been directed to an understanding of what occurs, as evidenced by careful clinical examination when the essential considerations in type and mode of exposure are appreciated. For example, an observation known but not usually expressed is that tremor, a feature of mercury poisoning by inhalation, is not present where these compounds have been rubbed into the skin. Neurosis is discussed not only in relation to such conditions as miner's nystagmus and writer's cramp but also as the cause of lost time from work and in human relations generally. Illustrations of processes and medical findings are numerous and helpful except for some of the chest x-rays.

The bibliography is representative, most conveniently listed by subject at the end of each chapter. The index is more useful for the specific occupational diseases than for more general subjects; for example, rehabilitation, human relations, placement of workers, pneumonia, pneumonitis and pulmonary œdema are discussed but do not appear under these headings in the index.

This is more than a book of 1,000 pages for reference on occupational diseases. It should be required reading for medical students and should be in the hands of all general practitioners in industrial communities. It establishes, in an authoritative and interesting manner, a point of view toward the effect of occupational environment on health which every physician should have.

MEDICAL HISTORY OF THE SECOND WORLD WAR. THE ARMY MEDICAL SERVICES

Administration. Volume II. Edited by F. A. E. Crew. 564 pp. Illust. 65/-. Her Majesty's Stationery Office, London, England, 1955.

This is another volume in the United Kingdom series on the history of World War II, compiled under the direction of an editorial board and edited by Professor F. A. E. Crew. It is the second and last volume dealing with the administration of the Army Medical Services and concerned with the organizational development of certain of the components of the Army Medical Services. It thus provides the means of assessing the quality of their functioning. The book has 12 chapters and is adequately illustrated.

Chapter one traces the development and records the efficient and self-sacrificing work of the Army Nursing Service. There follow four chapters on the Army Hygiene Service. This Service records achievements in safeguarding the health of the soldier—a very notable contribution to the conservation of manpower. It records the work of the Hygiene Directorate, supplies, environmental hygiene and special hygiene problems such as airborne forces, armoured vehicle crews and divers. Included also is a chapter on certain diseases of military importance—infectious hepatitis, serum jaundice, malaria, scabies, scrub typhus, venereal disease, etc.—and the hygiene of prisoners-of-war, as it developed in extent and efficiency in all theatres of war.

Other interesting and well-written chapters are on the creation and evolution during the war of the Army Dental Service, the Army Pathology Service, the Army Blood Transfusion Service, the Army Psychiatric Service, the Directorate of Medical Research, the Army Ophthalmic Service, and the Army Radiological Service. A specially valuable and interesting chapter traces the development of the medical care of the Auxiliary Territorial Service, whose many problems were, from the point of view of the Army, entirely new and novel.

The judicious selection of material, no doubt from an avalanche of documents and reports, the clarity of presentation and the absence of didactic dogmatism are a few of the features that make this book outstanding. These historical accounts teach lessons from the past which should be of value for the future.

THE SURGERY OF THEODORIC ca. 1267 A.D.

Translated from the Latin by Eldridge Campbell and James Colton. Issued under the auspices of the New York Academy of Medicine. 223 pp. Illust. $5.50. Appleton-Century-Crofts, Inc., New York, 1955.

This admirable translation of the first two parts of a work originally published in four books in 1267 A.D. is published by Appleton-Century-Crofts Inc. for the New York Academy of Medicine. It is a most interesting addition to our knowledge of surgery as practised in the Middle Ages.

Theodoric, 1205-1298, was perhaps the son but certainly the pupil of Hugo of Lucca, 1160-1257. His own famous pupil was Henri de Mondeville, 1260-1320. Hugo left no published record of his work but Theodoric enthusiastically extols the work and teaching of his master.

For centuries before and after the time of Theodoric the doctrine of "laudable pus" had been taught and practised. Hugo and Theodoric advocated the desirability and described a method of attaining healing of wounds by first intention. This teaching went unheeded for centuries and was revived by Lister and MacEwen. The method of Hugo and Theodoric was control of hæmorrhage and cleansing of all wounds by sponging with hot wine. The edges of the wound were trimmed and sutures applied. Venous hæmorrhage was managed by pressure, and arterial bleeding by ligature of the vessel above and below the opening and division of the artery between the ties. Compound fractures of the skull were treated by elevation of fragments and the removal of loose pieces. Extradural hæmatomata were evacuated and all scalp wounds closed by sutures. The extraction of arrow heads was a major problem. The late W. W. Keen of Philadelphia and the late William Burt of Paris, Ontario, were probably the surgeons latest in time to encounter cases of arrow injury on this continent.

This book is fascinating reading for the student of the history of medicine and should be interesting to practising surgeons. The second half of the treatise is in process of translation and will be eagerly looked for. The translators have earned a rich reward of gratitude from the profession.

DOCTORS IN THE SKY

The Story of the Aero Medical Association.

R. J. Benford, Colonel, Medical Corps, United States Air Force. 326 pp. Illust. $9.50. Charles C Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1955.

This is chiefly a record in chronological order of the events, and of the individuals concerned with them, leading to the formation of the Aero Medical Association of the United States and its development to the present time. While the last four words of the title of the Association were dropped in 1947, the events and personalities depicted are chiefly "south of the border". The value of the book is historical. It is unlikely to have a wide distribution and is largely of interest to those concerned with aviation medicine.

The Association has devoted itself to assisting the progress of both military and commercial aviation. A description of the annual meetings from 1929 until 1955 is given. There are some excellent photographs, chiefly of individuals who have held important posts in the Association. The book also contains a number of appendices. There is a list of those physicians certified in Aviation Medicine by the American Board of Preventive Medicine. Another appendix contains a selected bookshelf of aviation medicine, which would be of great value to anyone with particular interest in the subject.

In summary, this book is not for the casual reader.